IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: COR 2010 001571

FINDING INTO DEATH WITH INQUEST

Form 37 Rule 60(1)

Section 67 of the Coroners Act 2008

Inquest into the Death of: Constandia PETZIERIDES

Delivered On:	5 June 2014
Delivered At:	Coroners Court of Victoria Level 11, 222 Exhibition Street Melbourne 3000
Hearing Dates:	22 April 2013 21 August 2013
Findings of:	Coroner Paresa Antoniadis SPANOS
Representation:	Mr Jim STAVRIS appeared on behalf of the family of the deceased. Mr Paul HALLEY of Counsel, instructed by Ms Mia CAMPBELL of Avant Law, appeared on behalf of Dr Chez Smith. Mr Sean CASH of Counsel, instructed by Mr Jayr TENG of Western Health, appeared on behalf of Western Health.
Police Coronial Support Unit:	Leading Senior Constable Tania CRISTIANO, assisting the Coroner.

I, PARESA ANTONIADIS SPANOS, Coroner,
having investigated the death of CONSTANDIA PETZIERIDES

and having held an inquest in relation to this death at Melbourne on 22 April and 21 August 2013:

find that the identity of the deceased was CONSTANDIA (also known as CONNIE) PETZIERIDES

born on 26 January 1936, aged 74

and that the death occurred on 27 April 2010

at 9 Sedgefield Terrace, Cairnlea, Victoria 3023

from:

I (a) HAEMOTHORAX
I (b) DISSECTING THORACIC AORTA.

in the following circumstances:

BACKGROUND & PERSONAL CIRCUMSTANCES

1. Mrs Petzierides was a 74-year-old married and retired woman who resided with her husband at the above address. Mrs Petzierides enjoyed reasonable health for a woman of her age and was independent with all daily living activities. In the period immediately preceding her death, Mrs Petzierides had been well with no acute medical problems. She was taking a number of prescription medications for a number of medical conditions, with no recent changes to her medication regime.

MEDICAL HISTORY

2. Dr Mark Hobart was Mrs Petzierides' regular general practitioner (GP) from May 1987 until his last consultation with her on 18 March 2010. According to Dr Hobart, her medical history included hypertension (1977), right renal cyst drainage (1997), gout (2001), left vitreous detachment (2002), osteoarthritis of both knees (2003) and left sciatica (2005).[1] Cardiac investigations were undertaken by Cardiologist Dr Deepak Haikerwal in December 2006, ahead of a left total knee replacement in January 2007. An echocardiogram was reported as essentially normal, except for moderately dilated left atrium and mild left ventricular hypertrophy, a coronary angiogram demonstrated mild to moderate coronary

[1] Exhibit A (Dr Hobart's statement dated 25 January 2013) and transcript pages 6-7.

artery disease with normal left ventricular systolic function, and an electrocardiogram (ECG) demonstrated previously unknown atrial fibrillation.[2]

3. Following her total knee replacement, the diagnosis of atrial fibrillation was confirmed, treatment with warfarin was initiated by Dr Haikerwal, and monitored on an ongoing basis by her GP with regular INR testing and dosing adjustments by a pathology service. According to Dr Hobart, Mrs Petzierides' warfarin therapy was reasonably stable, but her blood pressure had been difficult to control in the past and although it was generally well-controlled, could fluctuate.[3] Other than the lability of her blood pressure, Dr Hobart testified that he had no grounds for concern about her cardiac function or the possibility of any vascular disease.[4]

4. As at April 2010, Mrs Petzierides' regular (relevant) medications were Coumadin,[5] Progout,[6] Felodur ER,[7] Noten,[8] Micardis[9] and Panamax when required for pain relief.

THE COURSE OF MRS PETZIERIDES' FINAL ILLNESS[10]

5. Shortly after midnight on 26 April 2010, Mrs Petzierides woke with sharp left scapular pain radiating to both jaws, with associated nausea and sweating. At 0015hrs on 27 April 2010, her husband and son called 000 requesting an ambulance.

6. An ambulance was dispatched at 0016hrs (Code 1/Time critical) and arrived at 0022hrs. Mrs Petzierides complained of left scapular pain, described as a tight feeling, and head pain also described as a tight feeling. She gave a past history of gout and hypertension and her current medications. On secondary survey, Mrs Petzierides complained of nausea, 'pain described as tightness and left thoracic region and around jaw, radiating to head, denies aggravation'. She had good grips bilaterally. Her chest was clear with no signs of

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[2] Exhibit A and transcript page 7.

[3] Transcript pages 8-9.

[4] Transcript page 11.

[5] A brand name for warfarin, an anticoagulant drug.

[6] A brand name for allopurinol, a drug used to prevent gout.

[7] A brand name for felodipine, a calcium channel blocker vasodilator and antihypertensive drug.

[8] A brand name for atenolol, a beta-blocker, anti-arrhythmic, anti-angina and anti-hypertensive drug.

[9] A brand name for telmisartan, an angiotensin II receptor blocker vasodilator and antihypertensive.

[10] What follows in paragraphs 5 to 15 is a summary of events on 27 April 2010 which are largely uncontroversial. The clinical management and care provided to Mrs Petzierides in the Emergency Department of Western Hospital are discussed in more detail below from paragraphs 28 onwards.

respiratory distress, and ECG monitor revealed a heart rate of 42, uncontrolled atrial fibrillation and T wave inversion in Leads I, II and III and her blood pressure was 140/90.

7. The initial assessment of the ambulance paramedics was of ischaemic chest pain, and queried the possibility of aneurysm and/or thoracic dissection.[11] By way of treatment, they commenced oxygen therapy, set up an IV, withheld aspirin because of the suspicion of a possible thoracic aneurysm,[12] and called for MICA assistance.

8. The MICA crew arrived at 0030hrs, received a handover of the patient and immediately took over her care. They elicited a past history of hypertension and atrial fibrillation. In terms of the presenting complaint, they documented that at midnight, while going to bed, Mrs Petzierides experienced sudden onset 10/10 left scapular pain, referring to the jaw, described as sharp and heavy ('hasn't had before'),[13] with associated nausea, and one episode of vomiting in the presence of ambulance paramedics. They assessed Mrs Petzierides' respiratory status as normal, and ECG indicated a heart rate of 48, controlled atrial fibrillation and T wave inversion in Lead I.

9. Their initial assessment was documented as 'acute coronary syndrome NSTEMI >> For investigation', as was their final assessment.[14] It is likely that the possibility of aortic dissection formed part of the handover between the ambulance and the MICA paramedics. In any event, their treatment of Mrs Petzierides is clearly mindful of this possibility. They took bilateral blood pressures and assessed bilateral limb perfusion,[15] and did not administer aspirin until 0039 after 'further questioning re potential aortic involvement'.[16] The aspirin appeared to the MICA paramedics to be of no effect,[17] but Mrs Petzierides' nausea appeared to respond to metoclopramide, with no further episodes of vomiting.

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[11] This is an interpretation of the relevant part of the VACIS electronic Patient Care Report (at pages 38-42 of Exhibit J the coronial brief). Verbatim, the entry reads "ischaemic chest pain; ???aneurysm (thorasic) dissection".

[12] Ibid, again an interpretation of "0027 Withheld – Aspirin >> ?? possible thorasic aneurism".

[13] It is unclear if this refers to the strength of the pain or the type or quality of pain, or both.

[14] The VACIS electronic Patient Care Report made by the MICA paramedics was provided to the receiving hospital on admission to the Emergency Department and formed part of Mrs Petzierides' medical records (pages 32-37 of Exhibit J).

[15] The reasonable inference is that no significant discrepancy between the left and right arm was found and therefore nothing to reinforce or give rise to a concern about aortic dissection.

[16] The VACIS report is silent as to what questions were asked and answers given, but a reasonable inference is that the MICA crew were satisfied that it was safe to administer the aspirin.

[17] I assume this to mean as an analgesic.

10. Mrs Petzierides' pain responded to morphine administered in 2.5mg doses intravenously at 0036hrs, 0041hrs, and en route at 0048hrs. A further 2.5mg was administered en route at 0053hrs. Initially, this appeared to settle Mrs Petzierides' pain, before a significant increase in her pain shortly before arrival at the Emergency Department of Western Hospital (ED) when 5mg morphine was administered with no effect.[18] As evidenced by the cardiac monitor, Mrs Petzierides' blood pressure and cardiac function remained stable en route, but she developed increasing dizziness.[19]

11. Mrs Petzierides arrived in the ED at 0104hrs and was triaged within a few minutes of arrival.[20] The triage nurse noted 'chest pain into jaw, originates in shoulder blade, feels like a sharp pressure, pain score down from 5 [out of 10] to 2 with GTN. Now patient complains of pain all over', took observations[21] and allocated Mrs Petzierides triage category 2, with a target assessment medical officer within 10 minutes.[22] It was uncontroversial at inquest that the allocation of Mrs Petzierides to triage category 2 was appropriate.

12. Medical assessment of Mrs Petzierides did not take place until 0250hrs[23] when she was seen by Dr Chez Smith, an Emergency Medicine Registrar of some ten weeks' experience. The clinical management and care provided to Mrs Petzierides in the ED will be discussed in detail below, as this was the primary focus of the coronial investigation of her death. Suffice for present purposes to say that Dr Smith made differential diagnoses of myocardial infarction, unstable angina, pneumothorax or musculoskeletal pain. He ordered investigations in the form of a full blood examination, a chest x-ray and serial cardiac enzyme testing.

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[18] The VACIS report documents her pain scores as follows – 0032 10 of 10 (before any analgesia); 0036 10 of 10 (morphine 2.5mg , pain "a bit better"); 0041 8-severe (morphine 2.5mg, pain decreased to 2/10); 0048 2 – mild; 0053 1 of 10 (morphine 2/5mg, patient settled then significant increase in pain prior to arrival); 0102 8 – severe (morphine 5mg no effect, patient states pain increasing, clearly uncomfortable, irritable, stating pain severe). Exhibit J pages 35-36.

[19] Blood pressure readings documented on the VACIS report were 0032 130/80; 0036 140/80; 0041 140/80; 0048 137/80; 0053 130/80; 0102 135/80. Exhibit J page 36.

[20] According to the MICA crew's VACIS report, arrival was at 0104 and triage at 0108 (Exhibit J page 33). The Western Hospital medical records bear a time stamp of 0107 attributable to triage (Exhibit J page 89), and an admission time 0110 (Exhibit J page 87). The discrepancy is immaterial.

[21] The triage nurse was RN Paula Jane D'Alterio who was not required to testify at inquest. Her statement dated 23 December 2010 is at pages 8-9, 49 of Exhibit J. RN D'Alterio documented the following "triage" observations – respiratory rate 22, oxygen saturations 98% on 8 litres of oxygen/minute, radial pulse 52 beats per minute, strong, regular and beta blocked, temperature 36.7, nil distress.

[22] According to the Australasian Triage Scale, reproduced at pages 50-52 of Exhibit J.

[23] This is significantly beyond the target time for triage category 2 but not explored in this investigation as "delayed" diagnosis did not appear to be an issue in the circumstances.

13. While Mrs Petzierides remained in the ED, nursing observations were made every 15-20 minutes between 0120hrs and 0718hrs. Apart from complaining of dizziness, for which she was administered ondansetron[24] with only partial respite, she remained stable and pain free, requiring no further analgesia. When repeat troponin levels taken at 0600hrs were normal (0.03), Mrs Petzierides was discharged home with a diagnosis of musculoskeletal pain and a discharge letter to her GP recommending referral for an ECG/stress test.[25]

14. Mrs Petzierides left the ED some time after 0730hrs and went home with her family. A few hours later, at about 1245hrs, she was found by family members collapsed and unresponsive, in the bathroom of her home. Emergency services were called and an ambulance dispatched at 1249hrs (Code 1/Time critical) arriving at 1255hrs.

15. The attending ambulance paramedics observed agonal respirations, no palpable pulse or blood pressure. They commenced cardiopulmonary resuscitation (CPR) to which Mrs Petzierides initially responded with spontaneous respiration and a weak pulse, but no recordable blood pressure. Despite the administration of adrenaline in increments, her blood pressure remained unrecordable and cardiac output was eventually lost. After some 62 minutes, paramedics ceased CPR, and reported the death to the police, who in turn reported the death to the Coroner.

PURPOSE OF A CORONIAL INVESTIGATION

16. Mrs Petzierides' death was reported to the Coroner on the basis that it was an unexpected death, as it clearly was. The purpose of a coronial investigation of a *reportable death*[26] is to ascertain, if possible, the identity of the deceased person, the cause of death and the circumstances in which death occurred.[27] The *cause* of death refers to the *medical* cause of death, incorporating where possible the *mode* or *mechanism* of death. For coronial purposes, the *circumstances* in which death occurred refers to the context or background and

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[24] An anti-emetic drug, marketed as "Zofran". Ondansetron is the only mediation documented in the medical records pertaining to his admission (Exhibit J page 101).

[25] Exhibit J pages 91, 93.

[26] The *Coroners Act 2008*, like its predecessor the *Coroners Act 1985*, requires certain deaths to be reported to the Coroner for investigation. Apart from a jurisdictional nexus with the State of Victoria, the definition of a reportable death in section 4 includes deaths that appear *to have been unexpected, unnatural or violent or to have resulted, directly or indirectly, from accident or injury* and the *death of a person who immediately before death was a patient within the meaning of the Mental Health Act 1986".*

[27] Section 67(1) of the *Coroners Act 2008*. All references which follow are to the provisions of this Act, unless otherwise stipulated.

surrounding circumstances, but is confined to those circumstances sufficiently proximate and causally relevant to the death, and not merely all circumstances which might form part of a narrative culminating in death.[28]

17. The broader purpose of any coronial investigations is to contribute to the reduction of the number of preventable deaths through the findings of the investigation and the making of recommendations by coroners, generally referred to as the *prevention* role.[29]

18. Coroners are also empowered to report to the Attorney-General in relation to a death; to comment on any matter connected with the death they have investigated, including matters of public health or safety and the administration of justice; and to make recommendations to any Minister or public statutory authority on any matter connected with the death, including public health or safety or the administration of justice.[30] These are effectively the vehicles by which the prevention role can be advanced.[31]

19. It is important to stress that Coroners are not empowered to determine the guilt of any person, or the extent of any civil liability arising from a death.[32]

INVESTIGATION – SOURCES OF EVIDENCE

20. This finding is based on the totality of the material the product of the coronial investigation of Mrs Petzierides' death. That is the brief of evidence compiled by Leading Senior Constable Tania Cristiano from the Police Coronial Support Unit (PCSU), the statements, reports and testimony of those witnesses who testified at inquest and any documents tendered through them, and the final submissions of Counsel. All of this material, together

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[28] This is the effect of the authorities – see for example *Harmsworth v The State Coroner* [1989] VR 989; *Clancy* v *West* (Unreported 17/08/1994, Supreme Court of Victoria, Harper J.)

[29] The "prevention" role is now explicitly articulated in the Preamble and purposes of the Act, cf: the *Coroners Act 1985* where this role was generally accepted as "implicit".

[30] See sections 72(1), 67(3) and 72(2) regarding reports, comments and recommendations respectively.

[31] See also sections 73(1) and 72(5) which requires publication of coronial findings, comments and recommendations and responses respectively; section 72(3) and (4) which oblige the recipient of a coronial recommendation to respond within three months, specifying a statement of action which has or will be taken in relation to the recommendation.

[32] Section 69(1). A Coroner must not include in a finding or comment any statement that a person is, or may be, guilty of an offence. However, if a Coroner believes an indictable offence may have been committed in connection with a death, they must refer the matter to the Director of Public Prosecutions. Sections 49(1) and 69(2).

with the inquest transcript, will remain on the coronial file.[33] In writing this finding, I do not purport to summarise all the material and evidence, but will refer to it only in such detail as is warranted by its forensic significance and in the interests of narrative clarity.

21. In making comments and recommendations about matters connected with the death, pertaining to public health and safety, in furtherance of the Court's prevention role, I have also relied on an analysis of coronial data undertaken by the Coroner's Prevention Unit,[34] and on a Round-table meeting with Emergency Physicians held at the Court on 28 August 2013.[35] Both of these sources of "evidence" will be discussed below.

FINDINGS AS TO UNCONTENTIOUS MATTERS

22. In relation to Mrs Petzierides' death, most of the matters I am required to ascertain, if possible, were uncontentious from the outset. Her identity, the date, time and place were never at issue. I find, as a matter of formality, that Constandia Petzierides, known as Connie, born on 26 January 1936, aged 74, late of 9 Sedgefield Crescent, Cairnlea, Victoria 3023, died at her home at about 2.00pm on 27 April 2010.

THE MEDICAL CAUSE OF DEATH

23. Nor was the medical cause of death controversial. On 4 May 2010, an autopsy was performed by Forensic Pathologist Dr Paul Bedford, from the Victorian Institute of Forensic Medicine (VIFM), who also reviewed the circumstances as reported by the police to the Coroner, and post-mortem CT scanning of the whole body (PMCT), and provided a written report of his findings.

24. Dr Bedford found no significant traumatic injuries, and found evidence of a number of natural disease processes that were incidental to the cause of death. Of significance, he found a ruptured pericardial sac; a markedly enlarged heart (with no evidence of recent or remote infarction), and extensive haemorrhage around the posterior and basal aspects of the heart associated with a rupture of the aorta. There were two tears of the aorta - a posterior

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[33] From the commencement of the *Coroners Act 2008* (the Act), that is 1 November 2009, access to documents held by the Coroners Court of Victoria is governed by section 115 of the Act.

[34] A summary of this analysis appears under Comments in paragraph 1-6 below.

[35] Attachment 1 is a list of those who participated in the Round-table meeting. A summary of the consensus views is at paragraph 10 of the Comments section below.

inferior wall tear around the arch associated with haemorrhage into the tissues behind the aorta and into the left thorax, and a second horizontal tear just above the aortic valves measuring 35mm in length. There was 1500mL of blood in the left pleural cavity and 100mL in the right. Histology was in keeping with these macroscopic findings.[36]

23. In conclusion, Dr Bedford attributed death to *hemothorax* secondary to *dissecting thoracic aorta* and commented that the autopsy demonstrated extensive loss of blood into the chest cavity following a dissection of the aorta in the chest, and background heart disease with an enlarged and scarred heart.

24. Dr Bedford's finding of two discrete tears of the aorta at autopsy suggests that Mrs Petzierides may have experienced the pain of one tear immediately before the first call to 000 and her presentation to the ED, before experiencing the second tear about twelve hours later, that led to her catastrophic collapse and death. This hypothesis is in keeping with the known circumstances, and it is likely that rupture of the peridcardial sac was the terminal event.[37]

25. I find that the medical cause of Mrs Petzierides' death is haemothorax secondary to dissecting thoracic aorta.

FOCUS OF THE CORONIAL INVESTIGATION AND INQUEST

26. As foreshadowed above, the primary focus of the coronial investigation, including the inquest, was on the adequacy of the clinical management and care provided to Mrs Petzierides between her arrival in the ED shortly after 0100hrs on 27 April 2010, and her discharge home shortly after 0730hrs on the same day. Specifically, whether the diagnosis of aortic dissection should have been made by Dr Chez Smith, and if so, why it was not made.

27. This is a complex question that requires consideration of the symptoms and signs of aortic dissection, an assessment of Mrs Petzierides' clinical presentation against those symptoms and signs, consideration of Dr Smith's formal qualifications and experience and what could

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[36] Histology of autopsy samples, showed, inter alia "Moderate interstitial fibrosis ... in the heart with focal areas of increased fibroblast activity. In a left ventricular section there is a markedly narrowed mid calibre coronary artery. Moderate atheroma is noted in a direct coronary artery sample. There is moderate to marked myxoid degeneration in the aorta which is associated with some calcification. Dissection is confirmed..." Exhibit J page 6.

[37] Transcript page 88 and following where Dr Eddey expresses an opinion about this.

reasonably be expected of him. It also requires characterisation of the failure to diagnose. Was this a matter of individual professional competence, simply human error, albeit with tragic consequences? Or do the circumstances in which Mrs Petzierides died demonstrate broader systemic problems that may be remediable and/or amenable to prevention-focused comments and/or recommendations?

THE EVIDENCE OF DR SMITH

28. Dr Smith provided a statement dated 23 December 2010,[38] in which he conceded in the preamble that he had no direct recollection of his involvement in Mrs Petzierides' management and made his statement after reviewing the medical records. It follows that his statement is limited to matters documented in the medical records, either by himself or others, and any matters not documented, potentially even significant matters, are lost to the coronial investigation. Dr Smith also testified at inquest, clarifying and elaborating on his statement.[39]

29. Dr Smith graduated in 2005,[40] had been working at Western Hospital as an Emergency Medicine Registrar since February 2010, and so had only ten weeks experience in this role at the time he attended to Mrs Petzierides. He had worked in other medical roles in the ensuing period, but Western Hospital was his first role as an emergency registrar.[41] Dr Smith explained that he was not in training to become an emergency physician, but was what some would refer to as a "non-accredited emergency registrar". Generally, two registrars would be rostered overnight, and on 27 April 2010, he believes he would have been the more junior of the two. While he acknowledged that an on-call consultant would also have been available to him, he did not seek a consultation as he did not see the need.[42]

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[38] Exhibit B is Dr Smith's two-page statement dated 23 December 2010.

[39] Transcript pages 12-43.

[40] His formal qualifications are Bachelor of Medicine and Bachelor of Surgery 2005 – transcript page 12, 16.

[41] Transcript pages 16-17. *"So in 2005 to 2006 I completed my internship at the Mater Hospital in Brisbane. From 2006 until April 2007 I ... worked in occupational health performing workplace medicals and immigration medicals at a place called Health for Industry in Brisbane. From April 2007 to February 2008 I was employed by the Tweed Heads Hospital as a urology resident... from 2008 until January 2009 I was employed as a HMO3 surgical resident at the Royal Melbourne Hospital, and from February 2009 until July 2009 I was employed at first as a surgical resident in ENT; and then in the remaining term, the last term, I was employed as a trauma registrar at the Royal Melbourne Hospital..."* Also transcript page 38.

[42] Transcript page 18. See also Dr Bryant's evidence about staffing and a culture encouraging consultation at transcript pages 53-55.

At the date of the inquest, Dr Smith was a general practice registrar in Coffs Harbour, New South Wales.[43]

30. According to Dr Smith's statement and evidence at inquest, he noted the triage nurse's assessment, and the observations taken by the cubicle nurse, when he first attended to Mrs Petzierides at 0250hrs.[44] He documented a history of presenting complaint as sudden onset 10/10 left upper back/scapular pain, sharp, radiating to both jaws, associated with nausea and with no shortness of breath, no cough, no sweating, also with radiation to the occipital scalp. He documented a past medical history that included atrial fibrillation, hypertension, gout, osteoarthritis and a left total knee joint replacement, and current medications as warfarin and noten. From reviewing her past medical records, he noted that a coronary angiogram undertaken in 2006 revealed mild to moderate coronary artery disease.[45]

31. On examination, Dr Smith documented that Mrs Petzierides was a well looking lady with a Glasgow Coma Score (GCS)[46] of 15, an irregular heart rate of 54 beats per minute, non-elevated jugular venous pressure, dual heart sounds heard with no murmur, chest clear and point tenderness over the left medial scapula/paraspinals.[47]

32. In terms of investigations, Dr Smith noted that the ECG showed atrial fibrillation and old inferior/anterolateral ST changes. Blood tests revealed normal full blood count, sodium 140mmol/L (within the normal range), potassium 3.4mmol/L (slightly low, normal range 3.5-5.5mmol/L), urea 8.8mmol/L (slightly high, normal range 2.5-8.3mmol/L), creatinine

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[43] Transcript page 17.

[44] For details of the triage nurse's assessment, see paragraph 11 above. The cubicle nurse's initial assessment documented at 0120hrs are 'Temperature 35.8°C, HR 45-65/min, AF, BP 175/79mmHg, RR 14/min, SpO2 100% on 8L oxygen. Pink, warm, dry, feels dizzy. Pulse strong, irregular. Unable to use pain score. Says she has 'a little bit' of pain but less than previously. Chest clear. Drowsy. ECG. Bloods taken.' Also transcript page 22.

[45] I have paraphrased the history as documented by Dr Smith in the medical record under HOPC (see page 90 Exhibit J), in preference to Exhibit B which omits reference to "sudden onset 10/10", and transcript pages 13-14 where Dr Smith read his statement into evidence. While I do not suggest that the omission is necessarily colourable, this description of pain in these terms is probably significant, as will be discussed below. See also transcript page 26 where he acknowledges his notes in this regard.

[46] "A quick, practical and standardized for assessing the degree of conscious impairment in the critically ill and for predicting the duration and ultimate outcome of coma, primarily in patients with head injuries. The system involves three determinants: eye opening, verbal response and motor response, all of which are evaluated independently according to a rank order that indicates the level of consciousness and degree of dysfunction." Mosby's Medical Nursing and Allied Health Dictionary, Fourth edition, page 673.

[47] Exhibit J page 91. I note that while Dr Smith documented "BP, SpO2 and RR", he did not document any values against these parameters. In Exhibit B paragraph 11 (reiterated at transcript page 1) he adopts the nursing observations documented at 0235hrs and most contemporaneous with his examination - 'no pain, HR 51, BP 155/89, SpO2 99% on 8L oxygen.'

6.6umol/L (within the normal range), creatine kinase (CK) 180IU/L (elevated, normal range 20-160IU/L) and troponin I 0.02ug/L (within the normal range). In evidence, Dr Smith explained that although the cardiac enzyme CK was elevated, the normal troponin level was reassuring, as it is the more sensitive indicator of heart damage, and is replacing creatine kinase in the purposes of acute coronary syndrome risk stratification.[48] Mrs Petzierides' INR level was 2.3, high as regards the "normal" reference range (0.8-1.3), but appropriate for someone taking warfarin for atrial fibrillation, as she was, with no valvular disease.[49]

33. Although not documented as such in his notes, in his statement, Dr Smith indicates that his differential diagnoses were myocardial infarction, unstable angina, pneumothorax or musculoskeletal pain.[50] When a chest x-ray ordered and reviewed by him showed cardiomegaly and no pneumothorax, that diagnosis was excluded.[51]

34. In his statement, Dr Smith expanded on his notes, explaining his preference for a cardiac cause for Mrs Petzierides presentation in the following terms – *"On the basis of her existing cardiac history, her pre-existing coronary artery disease, hypertension, age, and the clinical features of the presentation she was risk stratified as intermediate-high risk for acute coronary syndrome".*[52] This informed his management plan which was for a period of observation in the ED, to await repeat troponin levels and, if they were normal, Mrs Petzierides was to be discharged home with a letter to her GP recommending referral for an ECG/stress test.[53]

35. He explained that he documented "musculoskeletal pain" under the heading "impression" in his notes, as it was the most likely diagnosis.[54] In both his statement and in evidence, Dr Smith recognised that musculoskeletal pain is a diagnosis of exclusion and should only be

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[48] Transcript pages 26-27. Note that a repeat troponin level taken at 0600hrs was 0.03ug/L, again with the normal range. See also Exhibit J at page 91,107-111 and Exhibit B at paragraph 13.

[49] Transcript page 27.

[50] Exhibit J page 91, Exhibit B paragraph 12.

[51] Transcript pages 27-29. Not unusually, Dr Smith needed to interpret the x-rays for himself, the formal radiologist's report at Exhibit J page 106 was likely unavailable during Mrs Petzierides episode of care in the ED. The report reads "Clinical Notes: Left-sided chest pain. Sudden onset. ?Pneumothorax. Past history of AF. Findings: The heart is markedly enlarged. Bibasal opacities which is likely contributed by soft tissue projections. There is no pneumothorax or diaphragmatic free gas."

[52] Exhibit B paragraph 13.

[53] Exhibit J page 92, transcript page 27.

[54] Transcript page 27.

made after excluding other likely life threatening diagnoses. However, he maintained that in this case he arrived at the diagnosis *based on the history of sharp left upper back, scapula pain radiating to the neck and the clinical examination findings of point tenderness over the left medial scapula/paraspinal region it the absence of abnormal investigations to suggest a cardiac, respiratory or vascular cause for the pain.*[55]

36. It has to be said that there is no explicit reference to aortic dissection in Dr Smith's notes, nor any unequivocal implicit indication that he considered the possibility at the time. Nevertheless, in his statement, Dr Smith maintained that he always considers a vascular cause such as ischaemia or aortic dissection in patients with trunk pain, and that in Mrs Petzierides he assessed the risk of aortic dissection as low, based on history, clinical examination and investigation results that did not demonstrate evidence of aortic dissection.

37. In particular, he relied on the chest x-ray that did not show a widened mediastinum, the fact that she was neither profoundly hypertensive nor hypotensive, the fact that (apart from initially having mild pain) she remained pain free within the ED and did not require any further analgesia.[56] At inquest, he reiterated that he did consider the possibility of aortic dissection in Mrs Petzierides, but incorrectly assessed her risk as low.[57]

38. At inquest, Dr Smith demonstrated appropriate professional reflection, having made a point of learning more about aortic dissection, and conceding that "in hindsight" the symptoms and signs were there to be seen, and actually supported an assessment that Mrs Petzierides was at high risk of aortic dissection, not low.[58]

39. The concept of hindsight is a familiar one in this jurisdiction, but is not always used in the same sense. In my view, what Dr Smith conceded in evidence, and to his credit appropriately, was that having learnt more about the variable presentations of aortic dissection, and the limitations of the investigations on which he relied, he accepted that the diagnosis was open, and should have been considered more assiduously. This is not hindsight in the sense that the diagnosis of aortic dissection could not be made at the time,

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[55] Exhibit B paragraph 15.

[56] Exhibit B paragraph 16.

[57] On behalf of the family, Mr Stavris cross-examined Dr Smith about whether and when he actually considered the diagnosis of aortic dissection, and he maintained that he considered the possibility on her arrival in the ED, but incorrectly assessed her as being at low risk - see transcript pages 33-34.

[58] Transcript pages 32-39 in particular.

given Mrs Petzierides clinical presentation and course while in the ED, but could only be invoked after the event, requiring the revelation of further symptoms or signs, a different clinical presentation or a clearer less ambiguous clinical course.

THE EVIDENCE OF OTHER EMERGENCY PHYSICIANS

40. In his capacity as Director of the Emergency Medicine Department of Western Hospital, Dr Michael Bryant provided a statement and testified at inquest.[59] Dr Bryant's evidence was that, in common with current emergency medicine practice, there is no specific guideline in the ED for the assessment of aortic dissection. Rather, medical staff are encouraged to consider differential diagnoses, and the relevant guideline or pathway pertains to undifferentiated chest pain, whether cardiac, respiratory, vascular or musculoskeletal in origin, and is prefaced by a reminder to consider differential diagnoses such as pulmonary embolism and thoracic aortic dissection.[60] The current guideline, tendered by way of example, only refers to aortic dissection is in the preamble as follows - ***"Please consider other diagnoses e.g. PE, dissecting thoracic aneurysm, etc. If these are possibilities, discuss with ED consultant or senior registrar as soon as possible to arrange investigation and management plan..."***[61]

41. Dr Bryant also gave evidence about postgraduate teaching in the ED, the provision of an ED Post Graduate Medical Education Orientation Manual (the Manual) prior to commencing a term within the ED, orientation to the ED including access to hard copy clinical pathways and electronic access to same. He stressed that one of the learning objectives identified in the Manual in respect of cardio/pulmonary emergencies were the differential diagnoses of myocardial infarction, pulmonary embolism, arrhythmias and aneurysm/dissection.[62]

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[59] Dr Bryant also participated in the Round-table meeting with Emergency Physicians referred to in paragraph 21 above and under Comments paragraphs 8-10 below. His statement (Exhibit D) was in response to a request to address the following issues – "Does your ED have guidelines for the assessment and management of patients presenting with chest pain for whom there is a significant suspicion of aortic dissection" If so, please provide the guideline. If not, do you consider a guideline would be helpful? What teaching do registrars and HMOs receive that is likely to improve recognition of the disease? Any further comments you would like to make?"

[60] Exhibit D page 1 and transcript page 44.

[61] Exhibit E was the version of the guideline in operation as at April 2013. It had not changed materially since 27 April 2010, that is as regards reference to thoracic aortic dissections. The guideline is designed for use in the Short Stay Unit, entitled "Rule Out Acute Coronary Syndrome Pathway" and, although not strictly relevant to Mrs Petzierides episode of care in the ED, Dr Bryant conceded that even after a short time in the ED staff become very familiar with the pathway and, effectively, follow it for patients treated outside the Short Stay Unit – transcript pages 47 and following.

[62] At page 6 of the Manual – see Exhibit E.

Registrars, including non-accredited emergency registrars like Dr Smith,[63] have equal access to education in the ED, including structured weekly education coordinated by the Director of Emergency Medicine – Training, that leads to Fellowship and Specialist recognition with the Australasian College of Emergency Medicine.[64]

42. As an experienced Emergency Physician, Dr Bryant was also in a position to give expert evidence about aortic dissections, albeit not as an independent expert, given his overall supervisory role of the ED. He testified that aortic dissection is a difficult diagnosis to make, that there is no validated clinical tool and no one sign or symptom that can positively identify an acute aortic dissection, and that, according to the literature, an estimated 38% of acute aortic dissections are missed on initial evaluation.

43. Dr Bryant suggested that diagnosis is confounded by the relative rarity of acute aortic dissection, compared with other causes of chest pain. For example, he testified that of about 33,000 ED presentations in the preceding year, more than 2,500 presented with chest pain. Of these 571 had an acute myocardial infarction, 235 were diagnosed with angina, 71 were diagnosed with pulmonary embolus and only two patients were diagnosed with an aneurysm, one of which was previously known.[65]

44. For all these impediments, Dr Bryant's evidence was that the diagnosis of acute aortic dissection is best made when there is a high clinical suspicion given the overall evaluation of the patient, including the history, physical examination, and supporting tests including ECG, laboratory studies and radiology. This presupposes a level of understanding of the pathology of acute aortic dissections, or an informed clinician. As Dr Bryant put it pithily, *"If the differential diagnosis of this condition is not considered then it is often missed."*[66]

45. Professor Anne-Maree Kelly is the Academic Head of the Emergency Medicine Department of Western Hospital. Prof Kelly provided clinical overview to Corporate Counsel for

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[63] This is confirmed in Prof Kelly's evidence at transcript page 120.

[64] Exhibit E, transcript page 54 and following. Also see Dr Kelly's evidence about training/education at pages 115-120.

[65] Transcript pages 50-51. This is a slightly different pathological process, but I understood that Dr Bryant was using the term interchangeably with aortic dissection. Prof Kelly also testified about the relative rarity of aortic dissection compared with other ED presentations.

[66] Exhibit E and transcript pages 55 and following.

Western Health, [67] and in due course was proffered as a witness at inquest. Prof Kelly is a senior Emergency Physician who has provided independent expert reports in other coronial investigations in this state, and elsewhere.[68] Her role in the investigation of Mrs Petzierides' death was necessarily less arm's length than that of an independent expert, but I accept that she has endeavoured to evaluate Dr Smith's clinical management fairly.

46. Without doing justice to her comprehensive clinical overview, in summary, Prof Kelly characterised Dr Smith's aim to rule out an acute coronary syndrome as sound, given that it is many times more common than aortic dissection and given Mrs Petzierides' known coronary artery disease. Prof Kelly did not find convincing evidence in the medical record, or in his own account, that Dr Smith had considered aortic dissection or aimed to exclude it as a diagnosis. She identified gaps in history taking and examination with respect to neurological features and blood pressure measurement, and, significantly, gaps in the logic stated by Dr Smith for excluding the diagnosis.[69]

47. According to Prof Kelly, Dr Smith's reliance on the absence of a widened mediastinum on chest x-ray, to exclude aortic dissection, was not sound. Although abnormalities of the aorta are seen in >80% of cases, 12-15% will have a normal chest X-ray, and so a normal chest x-ray cannot be safely used to exclude the diagnosis. The presence or absence of hypertension depends on the type of dissection, which in turn depends on the involvement of the ascending aorta, and is therefore also not an appropriate rule-out mechanism, as Type A dissections are often normotensive. In any event, in Mrs Petzierides, the presence or extent of any hypertension or tachycardia, would have been masked by her blood pressure and beta-blocking medications. Finally, while severe pain, classically described as unremitting, tearing or ripping in character, is the most common presenting symptom of aortic dissection,

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[67] Exhibit G was Prof Kelly's statement/report dated 26 February 2013, Exhibit H her Curriculum Vitae and Exhibit I Three Journal article on Aortic Dissection, namely (1) Braverman A C Acute Aortic Dissection: Clinician Update. Circulation 2010; 122:184-188. (2) Coman M "Aortic Dissection" in Cameron P, Jelinek G, Kelly Am, Murray L, Brown AFT. Textbook of Adult Emergency Medicine 3rd Ed. Chruchill Livingstone 2009. (3) Imamura H, Sekiguchi Y, Iwashita T et al. Painless acute aortic dissection- Diagnostic, prognostic and clinical implications. Circ J 2010 Dec 24: 75(1): 59-66.

[68] Transcript page 116-123 where Prof Kelly explains why it is difficult to diagnose aortic dissection. I note that Prof Kelly was also a participant in the Round-table meeting with Emergency Physicians referred to in paragraph 21 above and under Comments in paragraphs 8-10 below.

[69] Exhibit G and transcript pages 125 and following.

pain is maximal at onset and may resolve spontaneously, it may be migratory and sometimes aortic dissection may even be painless.[70]

48. At inquest, Prof Kelly explained that the diagnosis of aortic dissection was difficult to make, because the range of symptoms it presents is so broad, and the proportion of cases that present with the typical symptoms is so small. Prof Kelly testified that there is no diagnostic test that is 100 per cent accurate. Those tests that are available (CT aortography, transoesophageal echocardiography and MRI) involve significant cost, in terms of risks to the patient's health (from radiation exposure and contrast medium), the cost of providing the service and other resource implications (patient flow and displacement of other high-risk patients).[71] What is lacking, according to Prof Kelly, is a reliable tool for identifying those patients in whom the risks associated with these additional investigations, is outweighed by the risk that an aortic dissection is in unfolding.[72]

49. In respect of Mrs Petzierides, Prof Kelly expressed the opinion that her presentation was atypical for aortic dissection, but "with hindsight" there were some features that might point towards the diagnosis - pain in the back, radiation to jaw, severity of pain, an episode of hypotension in the ED[73] - all of which could be due to other causes. Significantly, the absence of neurological symptoms would point away from the diagnosis, as would the apparent failure by the ambulance crews to find a blood pressure difference between the arms, the description of pain as sharp, and the finding of local tenderness over the back muscles.[74] While Prof Kelly was somewhat troubled by the discharge diagnosis of musculoskeletal pain, for 10 out of 10 pain, she maintained that it was a reasonable differential diagnosis in the circumstances.[75]

50. While acknowledging some deficiencies, Prof Kelly's conclusion was that Dr Smith's clinical management did not fall short of the accepted standard of practice for a doctor of his

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[70] Exhibit G page 3 and Exhibit I.

[71] Transcript page 117-119. Exhibit G at page 4 "Other comments".

[72] Transcript page 118,124,132.

[73] Exhibit J page 93 where nursing observations note a BP of 92/67 AF slow at 0650hrs. See also transcript at pages 128-129,135-136.

[74] Exhibit G and transcript page 117, 121-122.

[75] See transcript page 143 where she speaks from her own experience of patients with "non-traumatic" or less obviously traumatic causes of back pain such as heavy gardening. Presumably, those patients gave a history of greater than usual exertion. It is not apparent that Mrs Petzierides was asked for or volunteered such a history.

training and experience, in circumstances where Mrs Petzierides' presentation was not entirely typical of aortic dissection, and would have challenged more experienced clinicians.[76]

INDEPENDENT EXPERT REPORT

51. Emergency Physician Dr David Eddey was the Director of Emergency Medicine at the Geelong Hospital since 1995. He was asked to provide independent expert report assessing the adequacy of the clinical management and care provided to Mrs Petzierides in the Western Hospital ED on 27 April 2010, by reference to current standards of medical and nursing practice; to conduct a literature review regarding the incidence of aortic dissection including rate per 100,000 of population and differential diagnoses and to advise as to world's best practice protocols.

52. He produced a comprehensive, scholarly yet accessible and practical 56-page report, salient excerpts from which are attached to this finding.[77] Aside from contributing to an assessment of the clinical management and care provided to Mrs Petzierides in the ED on 27 April 2010, the report was fundamental to the Round-table meeting with Emergency Physicians, in which he also participated.

53. That part of Dr Eddey's report in which he assessed the clinical management and care provided to Mrs Petzierides in the ED, effectively by Dr Smith, is attached to this finding.[78]

54. Dr Eddey identified the communication of information from AV paramedics to the ED as a clinical issue. He noted the likelihood that the suspicion of thoracic aortic dissection formed by the first ambulance crew, while it may have been conveyed verbally from the first ambulance crew to the MICA crew and by them to the triage nurse, did not appear in the medical records.[79] It may be significant that the first ambulance crew attended closest to the

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[76] I have paraphrased to some extent. This is my reading of her evidence as a whole. See Exhibit G page 4 and transcript pages 121-122 especially.

[77] Exhibit F was Dr Eddey's report dated 12 April 2013. Applications for a copy of the complete report may be made by Form 45 application, pursuant to section 115(3) of the Act accessible via the Court's website.

[78] "Attachment 1" to this finding is an excerpt from Dr Eddey's report – pages 45-50. It includes his analysis of the flow of information from the AV crews to the ED.

[79] It was common ground that current AV practice is for the transporting crew to provide their VACIS Patient Care Report to the receiving hospital, the reports of any paramedics who treated the patient earlier remain on their own VACIS tablets, and are effectively "lost" to clinicians during the patient's episode of care. See also "Attachment 1" pages 45-46, 48-49.

onset of pain and where best placed to elicit a contemporaneous description of the pain and/or to appreciate how unwell Mrs Petzierides was. Had their suspicions been conveyed to Dr Smith, it may have triggered consideration of the diagnosis of aortic dissection, or may have heightened his clinical suspicion for the diagnosis.[80]

55. In Dr Eddey's assessment, Dr Smith's reasoning that Mrs Petzierides was at low risk of aortic dissection was flawed on three bases –

1. The absence of mediastinal abnormality on chest x-ray should not be used to rule out aortic dissection, as 30-40% of dissections may have either normal chest x-ray or show no mediastinal widening or abnormal aortic outline.[81]

2. Normal blood pressure should not be used to rule out aortic dissection, as neither hypertension nor hypotension are sufficiently sensitive markers for the disease, and 40% of Type A dissections are likely to be normotensive.[82]

3. Dr Smith's comment that Mrs Petzierides only had mild initial pain the ED ignores the documented onset of the pain (both in terms of its sudden onset and severity),[83] and the fact that aortic dissection is a dynamic process than can occur in stages with pain initially settling, only to recur when the dissection recommences.[84]

56. Dr Eddey recognised that the clinical management and care provided to Mrs Petzierides followed a conventional chest pain pathway, directed primarily at ruling out myocardial ischaemia. Having come to the end of that pathway, without demonstrating any significant abnormalities, Mrs Petzierides was discharged home with a plan for cardiology review as an outpatient, with her presentation to the ED attributed to musculoskeletal pain.[85]

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[80] See related discussion at transcript pages 72-73, 138-140.

[81] Exhibit F pages 22-27 and "Attachment 1" page 48.

[82] Exhibit F page 26 and "Attachment 1" page 48.

[83] Inserted by way of explanation, and also ignores the fact of pre-hospital administration of significant analgesia (300mg aspirin – probably administered for its blood thinning properties - and 15mg morphine over 23 minutes – see Exhibit J page 35) Also, see transcript pages 76-77 where Dr Eddey gives evidence about characterisation of Mrs Petzierides' pain.

[84] "Attachment 1" pages 47-48 and transcript pages 91 and following.

[85] Exhibit F page 36 and "Attachment 1" page 36.

57. Dr Eddey expressed the opinion that despite a history of risk factors for aortic dissection and a description of the onset of pain that is highly suspicious, Dr Smith did not look for the classic clinical signs of aortic dissection. His assessment that Mrs Petzierides' risk of aortic dissection was low, was flawed, as it was based on an incomplete examination, incorrect information and flawed reasoning arising from a lack of awareness of the limitations of the criteria used to exclude the disease.[86]

58. While Dr Eddey also referred to the concept of hindsight, it is tolerably clear from a reading of his evidence in its totality, that he was not using the expression in its strict sense. He was not suggesting that the diagnosis could only have been made after the event, and could not have been made in the ED based on what was known about Mrs Petzierides.

59. Dr Eddey agreed with evidence of Dr Bryant and Prof Kelly that aortic dissection is a relatively rare disease in the context of the more common causes of chest pain in patients presenting to emergency department and/or otherwise difficult to diagnose. He cited the literature that suggested that correct diagnosis is made in as few as 15-45% of presentation on initial assessment and a diagnostic delay of more than 24 hours after hospitalisation occurs in up to 39% of cases.[87]

60. Like Dr Bryant and Professor Kelly, Dr Eddey bemoaned the lack of a validated tool for risk stratifying patients suspected of having aortic dissection.[88] However, based on the literature, he asserted that the predominant presenting symptom in acute aortic dissection is sudden onset severe chest or back pain, compared to the generally slower increase in intensity of pain associated with acute myocardial infarction, and that it is in careful history taking around the nature of the pain that gains can be made in correct diagnosis.[89] Among other publications, Dr Eddey referred to the 2010 American Heart Association Released Guidelines, that characterised as "high-risk" chest, back or abdominal pain that is abrupt or

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[86] "Attachment 1" page 49 and transcript pages 80 and following.

[87] Exhibit F page 19 and transcript pages 85 and following.

[88] Exhibit F pages 34-35.

[89] Exhibit F pages 11-14.

instantaneous in onset, severe in intensity and has a ripping, tearing, stabbing or sharp quality.[90]

61. In responding to a request for advice as to any systemic impediments to correct diagnosis of aortic dissection in the context of ED presentations, Dr Eddey provided a comprehensive analysis which is attached to this finding,[91] as well as the following convenient summary –

1. Clinical rarity and failure to think of the condition.

2. Poor quality history taking and failure to clarify the characteristics of the pain.

3. A perception that aortic dissection is unlikely if the classic signs of blood pressure differential, pulse deficit and widened mediastinum are absent.

4. An emphasis on much higher volume cardiac chest pain and 'chest pain' pathways that focus primarily on acute coronary syndromes without encouraging re-evaluation of the patient and diagnosis.

5. Conditions within emergency department and work practices that encourage patients to be moved on within short time frames.[92]

62. Finally, as requested, Dr Eddey provided advice as to ED best practice with respect to suspected aortic dissections, with a focus on improving the diagnosis and survival of patients with aortic dissection, and drafted a sample information sheet suitable for dissemination in EDs. For convenience, I attach that part of Dr Eddey's report and his Appendix 1 entitled "Recognising Aortic Dissection."[93]

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[90] Exhibit F page 20. See also pages 11-14 where various textbook definitions of the pain associated with aortic dissection are reproduced.

[91] "Attachment 2" is pages 28-36 of Exhibit F.

[92] Exhibit F and "Attachment 2" at page 36.

[93] "Attachment 3" is pages 37-39 and 51-52 (Appendix 1) of Exhibit F.

CONCLUSIONS

63. The standard of proof for coronial findings of fact is the civil standard of proof, on the balance of probabilities, with the *Briginshaw* gloss or explication.[94] The effect of the authorities is that Coroners should not make adverse findings against or comments about individuals, unless the evidence provides a comfortable level of satisfaction that their departure from the prevailing standards of their profession, has caused or contributed to the death under investigation.

64. While not an edifying conclusion, and one with real ramifications for patient safety, the weight of the available evidence does not support a finding that, in failing to diagnose aortic dissection, Dr Smith departed from the prevailing standards of his profession. The standard against which his assessment and treatment of Mrs Petzierides is to be judged, is that of an Emergency Registrar with his formal qualifications and experience, and without the benefit of hindsight, and I use that phrase in its strict sense.

65. Such a finding does not equate with endorsement of the diagnosis of musculoskeletal pain which was a wholly inadequate response to sudden onset, interscapular pain, assessed by the patient as 10 out of 10, and requiring significant analgesia, particularly in the absence of a history of trauma or significant exertion, either elicited or volunteered.[95]

66. Nevertheless, Mrs Petzierides' death was preventable in the sense that her presentation to the ED was a lost opportunity for diagnosis of a thoracic aortic dissection. It may not be helpful to think in term of "classic" features of a disease that is a classic mimicker of other conditions, and/or because, it is a dynamic process.[96] A condition precedent to correct diagnosis of Mrs Petzierides was management by a clinician who was well informed, as opposed to simply aware of aortic dissection, as a differential diagnosis for chest pain, cognisant of the signs and symptoms of the disease, including its variability over the course

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[94] *Briginshaw v Briginshaw (1938) 60 C.L.R. 336 esp at 362-363.* "The seriousness of an allegation made, the inherent unlikelihood of an occurrence of a given description, or the gravity of the consequences flowing from a particular finding, are considerations which must affect the answer to the question whether the issues had been proved to the reasonable satisfaction of the tribunal. In such matters "reasonable satisfaction" should not be produced by inexact proofs, indefinite testimony, or indirect inferences..."

[95] "Attachment 1" page 47. Also discussion at paragraph 46 above and Dr Bryant's evidence at transcript pages 55-56.

[96] Transcript page 122.

of the disease and depending on whether a Type A or Type B dissection was involved, and the limitations of investigations used to make or to exclude the diagnosis.

67. In finding that Mrs Petzierides' death was preventable, I am not disregarding the significant mortality associated with the disease, even when correctly diagnosed and treated in a timely manner, whether medically or surgically.[97] Regardless, her death was preventable in the sense that correct diagnosis, and the commencement of treatment before catastrophic rupture gave Mrs Petzierides a reasonable chance of surviving an otherwise lethal condition, whereas discharge home gave her little, if any, chance.[98]

COMMENTS

Pursuant to Section 67(3) of the ***Coroners Act 2008***, I make the following comment(s) connected with the death:

1. Given the circumstances in which Mrs Petzierides' death occurred, and based in part on my own prior experience of the frequency of aortic dissection as a cause of death in the coronial jurisdiction, I asked for assistance from the Coroners Prevention Unit (CPU).

2. Specifically, I asked CPU to identify recent deaths resulting from aortic dissection to identify potential missed opportunities for intervention, and to identify factors that may lead to improved patient outcomes, with particular emphasis on those deaths where there was a presentation to a health service proximate to death, and the diagnosis of aortic dissection was either not considered, or was actively excluded.

3. CPU reviewed coronial data in the National Coroners Information System (NCIS) for the three year period 2010-2012. They identified 137 deaths resulting from aortic dissection: 44 in 2010, 47 in 2011 and 46 in 2012. (I note, incidentally, that this rate of occurrence is broadly consistent with the evidence of all three Emergency Physicians who testified at inquest, and the literature.)

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[97] Exhibit F pages 25-27 where as part of Dr Eddey's summary of the features of aortic dissection he includes – Treatment: Type A Medical treatment +/- replacement of proximal aorta +/- aortic valve +/- CABG. Type B Medical treatment +/- surgical repair or endovascular stenting if complicated. Outcomes: Medical treatment only Type A 90% mortality, Type B 11% mortality. Surgical treatment Type A 15-35% mortality, Type B 10-30% mortality (noting that indications for surgical intervention in Type B increase mortality).

[98] "Attachment 1" pages 49-50. Also Dr Kelly's evidence at transcript page 123.

4. Of these 137 deaths, there were 19 cases where there was evidence in the medical examiner's report,[99] or in the police report of death to the coroner,[100] that the deceased had presented to a health service with symptoms of ill health proximate to death.[101] Two had presented on the day of death, six the day before, nine between two and ten days before death, and two, more than ten days before death.

5. In terms of clinical presentation, of the same 19 cases, chest pain was a feature in 11 cases, back pain in three cases, and chest and back pain in three cases. In only two cases was there no complaint of chest and/or back pain.

6. Aortic dissection was not diagnosed in any of the 19 cases. Three patients were discharged with a diagnosed of pinched nerve, one was discharged after exclusion of heart disease, and the reasons for discharge for the remainder were either 'unknown' (7), 'not applicable' (7) or 'not specified' (1).[102]

7. I inferred that there was potential here to make a contribution to a reduction in the number of preventable deaths, by investigating Mrs Petzierides' death in the context of this data, with a focus on any systemic issues or impediments to the diagnosis of aortic dissection on presentation to emergency departments.

8. The CPU report, the salient features of which I have endeavoured to summarise above, and Dr Eddey's report, were provided to a number of Emergency Physicians across a number of Health Services in metropolitan Melbourne and Geelong, with an invitation from CPU, on my behalf, to participate in a Round-table meeting. The purpose of the meeting was to examine the issues of increasing awareness and detection of aortic dissection and to identify systems changes that could improve patient outcomes, with the aim of assisting the Coroner to provide evidence-based consensus comments and/or recommendations under the Act.

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[99] Generally a Forensic Pathologist from the Victorian Institute of Forensic Medicine (VIFM) will provide an "autopsy" report or an "inspection" report to the Coroner. While there is some variation in formulation between pathologists, haemopericardium secondary to ruptured aortic dissection/aortic dissection is not uncommon. Note that the now established practice at VIFM of routine post-mortem CT scanning of the whole body, leads to a situation that where evidence of aortic dissection is apparent, there may be no autopsy, particularly if the family objects to autopsy.

[100] Victoria Police Form 83 is the usual vehicle used by police to "report" a death to the Coroner. The quantity and quality of information it contains is very variable, as it is based on information immediately accessible to the attending police officer/s at the scene of death.

[101] Due to limitations inherent in the data set, and the likelihood that other relevant deaths were not reported to the Coroner, as a Death Certificate was provided by a treating medical practitioner, or otherwise, this is likely to be an underestimate of relevant deaths.

[102] This may be a limitation in the data. Perusal of the actual coronial files may have yielded more detail here.

9. The Round-table meeting took place at the Court on 28 August 2013. The meeting ran for some two and a half hours and was scribed but not recorded. All participants, myself included, sat around the bar table in Court 1. There was no formal agenda or structure, but some deft facilitation of discussion. A list of participants is attached.[103]

10. Frank and open discussion was encouraged and several consensus views became apparent:[104]

- There is an imperative for clinicians not to miss the vastly more common causes of chest pain, i.e. ischaemic heart disease.
- While there was recognition that a diagnosis of aortic dissection is excluded by CT scan of the chest, CT scanning is not always available, and is potentially harmful due to radiation dosage predisposing to cancer later in life, and adverse reactions to contrast media. Participants articulated that adopting a practice of performing more CT scans to exclude aortic dissection might improve the detection rate, but might also be counter-productive in producing more long term harm, given the rarity of the disease.
- One of the difficulties with diagnosing aortic dissection is that patients often present without the classical features, making correct diagnosis difficult, even for experienced clinicians. However, participants also agreed that sometimes the diagnosis is simply not considered, and it is this latter group where participants identified preventative measures as being of particular value.
- Widespread use of chest pain and acute coronary syndrome pathways of care in EDs might be adversely affecting the ability to detect aortic dissection. There was agreement on the importance of exclusion of other serious diseases including aortic dissection, both at the beginning, and at the end of such pathways.
- The implementation of time-based performance targets might affect the care of patients with aortic dissection and other uncommon diseases. Participants agreed that there was potential for greater use of short-stay medicine wards to allow more time for diagnostic consideration and investigation for such patients.

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[103] "Attachment 4" may be inaccurate as attendances were not formally taken and the list has been compiled from the original invitation list and collective memory. There were some last minute changes/delegations. Apologies to anyone who has been inadvertently omitted.

[104] A report on the Round-table meeting written by Prof George Jelinek and others has been submitted to the *Medical Journal of Australia* for publication. It is entitled "Closer cooperation between the Coroner and Emergency Physicians: collaboration to improve outcomes from aortic dissection." A final decision about publication in the MJA is pending.

- The value of focused history-taking was considered paramount. Participants stated that risk factors should be elicited and history-taking should focus on the onset, severity and nature of the pain.
- Investigating any blood pressure or pulse differentials between arms was suggested as an easy non-invasive investigation which participants acknowledged was an indicator of aortic dissection but not sensitive enough to be used to rule-out the diagnosis.
- Emergency medicine teaching could encourage a mantra that aortic dissection is to chest pain, what subarachnoid haemorrhage is to headache. Both are lethal conditions if not diagnosed and treated urgently, and the gains that have been made in the detection of subarachnoid haemorrhage recently, could be duplicated with respect to aortic dissection. Participants suggested that junior staff in EDs could be encouraged to present cases by reporting any 'red flags' for aortic dissection at the outset so that decisions regarding CT scanning could be made early.
- Further research was suggested, particularly aimed at developing a risk score for aortic dissection incorporating the main clinical features (history, examination findings, chest x-ray, d-Dimer),[105] where a certain score prompts CT scanning. Participants thought that that the Department of Health's Emergency Clinical Network might be able to assist in development and validation of such a score, which could be sensitive enough for clinical 'rule out' of a significant proportion of cases. The availability of such a tool would also be expected to assist in encouraging more frequent consideration of aortic dissection.

RECOMMENDATIONS

Pursuant to section 72(2) of the **Coroners Act 2008**, I make the following recommendation(s) connected with the death:

1. That the Australasian College for Emergency Medicine (ACEM) considers highlighting in training curricula the importance of considering the diagnosis of aortic dissection for patients presenting with chest pain, and the nuanced presentations of aortic dissection. This is particularly important where ED patients are treated in accordance with a chest pain pathway, and ischaemic heart disease has been excluded by appropriate testing. A practice

[105] Although d-Dimer testing was discussed at the Round-table, there was no consensus view about its value in aortic dissection. Since the Round-table, I have been advised of a published study that concludes that a negative d-Dimer is reliable in identifying those patients who do not require imaging – Shimony A, Filion KB, Mottillo S et al. "Meta-analysis of usefulness of d-Dimer to diagnose acute aortic dissection." American Journal of Cardiology 2011; 107:1227-1234.

of re-visiting the diagnosis at the end of the pathway and/or review by a senior clinician before discharge would improve patient safety.

2. That the Minister for Health, the Secretary of the Department of Health and/or the Departments Emergency Care Improvement and Innovation Clinical Network (ECIICN) consider funding research aimed at developing and evaluating a structured clinical tool for risk stratification of patients presenting with chest pain and suspected of having aortic dissection.[106]

3. That Ambulance Victoria investigates the feasibility of providing the receiving hospital with all VACIS Patient Care Reports pertaining to the patient's episode of care, so that important clinical information, including in particular the first responders' clinical impression, is available to inform the clinical management and care provided by hospital clinicians.

ACKNOWLEDGEMENTS

1. It would be remiss of me not to acknowledge and thank a number of people who made significant contributions to this coronial investigation. Firstly, the Petzierides family, who in their grief, embraced the idea that some good might come of a prevention focused investigation of the death of their wife and mother, and displayed generosity of spirit, patience and trust throughout the process.

2. I wish to formally record my thanks to Dr David Eddey for his comprehensive response to my request for an independent expert report, in my experience in this jurisdiction, unprecedented for the breadth and depth of his analysis.

3. My thanks to Dr Lyndal Bugeja, Manager of CPU, for driving production of the CPU report, to Ruth Bergman for seamless organisation of the Round-table meeting, and to Professor George Jelinek and Dr Sandra Neate, the Court's/CPU's in-house Emergency Physicians for lending their imprimatur to the Round-table meeting and facilitating discussion.

4. Finally, I wish to thank all those Emergency Physicians and others, who gave generously of their time and participated in the Round-table meeting, overcoming any trepidation they may have felt at engaging with the Court in this way, and contributing to the discussion with frankness and collegiality.

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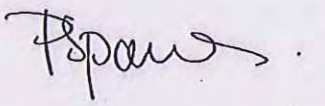
[106] See footnote 105 for discussion of the value of d-Dimer testing.

I direct that a copy of this finding be provided to the following:

- The family of Constandia (Connie) Petzierides
- Dr Chez Smith c/o Ms Mia Campbell, Avant Law
- Western Health c/o Mr Jayr Teng, Corporate Counsel, Western Health
- Dr Michael Bryant, Director of Emergency Medicine, Western Hospital
- Professor Anne-Maree Kelly, Joseph Epstein Centre for Emergency Medicine Research
- Dr David Eddey, Director of Emergency Medicine, Geelong Hospital
- Australasian College for Emergency Medicine
- The Honourable David Davis, Minister for Health
- Dr Pradeep Philip, Secretary, Department of Health
- Ms Jan Pannifex, Program Manager, Emergency Care Improvement and Innovation Clinical, Department of Health .
- Mr Colin Grant, Ambulance Victoria
- Leading Senior Constable Tania Cristiano, Police Coronial Support Unit
- The participants of the Round-table meeting.

Signature:



PARESA ANTONIADIS SPANOS
Coroner
Date: 5 June 2014

In the Coroners Court of
Victoria at Melbourne

Court Reference: COR 2010 001571

Inquest into the death of **Constandia PETZIERIDES**

Attachment 1

Assessment of clinical management provided at Western Health.

The medical care of Mrs PETZIERIDES has commenced with triage at 1:07. The triage nurse assigns at Category 2 (ideally seen within 10 mins). This is an appropriate category. The triage notes do not record the mode of onset of the pain, nor do they note a pain score of >5, or the concern of the AV crews regarding aortic dissection.

The 'cubicle' nursing notes record a history of: *"12 midnight sudden onset left scapular pain radiating to jaw – stabbing, pressure"*. Mrs PIETZERIDES pain resolves over this time. There is no reference to the severity of the pain or reference to Ambulance Victoria case notes.

Mrs PIETZERIDES is seen by Dr SMITH approximately 1 hour and 45 mins after arrival. By this time she is pain free. He conducts a history and examination and orders appropriate investigations. Dr SMITH obtains a clear history *of "sudden onset 10/10 left upper back/scapula pain – sharp – radiating to both jaws with nausea."*

ECG, Chest x-ray and blood tests are ordered.

Management has subsequently been directed at risk stratifying the patient for ischemic heart disease. Dr SMITH indicates in his statement that he considered Mrs PIETZERIDES to be at intermediate to high risk for ACS. Mrs PIETZERIDES was kept in the ED to have a 6 hour troponin measured and was discharged home with a plan for further cardiac assessment as an outpatient and with a diagnosis of musculo-skeletal pain, based upon the finding of point tenderness over the left scapula.

Dr SMITH does not appear to have considered admitting Mrs PIETZERIDES to the short stay unit. The Western Hospital Short Stay Unit Chest Pain guideline specifically asks clinicians to consider other diagnoses, including dissecting thoracic aneurysm and if *"these are possibilities, discuss with ED consultant or senior registrar as soon as possible to arrange investigation and management plan"*. A clinician admitting Mrs PIETZERIDES to the Short Stay Unit may have acted on this, but it is possible that the possibility would have been discounted, as did Dr SMITH, and no further action taken. As with other chest pain pathways or guidelines, this guideline provides no assistance to clinicians with regard to information on high-risk symptoms or signs.

Clinical Issues Identified

1. Communication of Ambulance information.

It is likely that the clinical impression/differential diagnosis (ie *"Ischaemic chest pain, aneurysm (thoracic) dissection"*) formed by the first AV crew to attend Mrs PETZIERIDES was not communicated to the hospital. As the original crew did not

transport the patient to hospital, their case notes (stored on their 'VACIS' tablet) would not have accompanied Mrs PETZIERIDES to the hospital.

There are no contemporaneously printed case notes from the first AV crew in the material provided, and the case notes in the file have been printed for the investigation of this matter on 20/12/2012.

The MICA crew transported Mrs PETZIERIDES to hospital. The only reference to the aorta in the case notes of this crew is found 'buried' in the clinical observations and notes where there is a record of consideration being given to the withholding of aspirin because of concern regarding "aortic involvement". The working diagnosis or final assessment recorded by the MICA crew on the case notes printed at 01:55 on 27 April 2010 was *"Acute coronary syndrome/NSTEMI"*. These notes would have been available to ED staff.

Whilst it is possible that the first attending crew or the MICA crews concern regarding aortic dissection was communicated to the triage nurse at Western Hospital verbally, there is no indication in the triage notes or any other hospital notes that this concern was raised by the MICA crew. It is unlikely that Dr SMITH spoke to the crew directly, as it is recorded that he attended the patient almost 1 hour and 45 mins after arrival.

It is conceivable that if hospital staff had been aware of the concern of the first ambulance crew to attend Mrs PETZIERIDES their notes, history, examination and investigation and management plan may have reflected this.

It is notable that the first Ambulance Victoria crew to attend Mrs PETZIERIDES are the only clinicians to directly consider aortic dissection as a differential diagnosis and to specifically perform any examination directed at looking for evidence of it..

2. Diagnosis decision making.

It is clear that Mrs PETZIERIDES experienced sudden onset of severe chest pain prior to attending hospital. Unfortunately her symptoms have not sufficiently raised a level of clinical suspicion in medical or nursing staff for them to consider pursuing the cause of the pain other than the consideration of ischemic chest pain. With the benefit of hindsight it was unsafe to dismiss this presentation as musculo-skeletal pain, once an acute coronary syndrome was thought to be unlikely.

There is nothing in the medical or nursing notes to indicate that aortic dissection was specifically considered as a differential diagnosis or looked for. Dr SMITH's clinical impression is recorded as "musculoskeletal" pain in the notes, and although tests are performed looking for evidence of myocardial ischemia and thoracic causes such as a pneumothorax, there is no record in the notes of specific examination of the patient

for key 'classic' clinical signs (widened mediastinum, abnormal aortic outline, blood pressure difference or pulse deficits), although this may have been done.

I am not aware of a non-traumatic musculoskeletal condition giving rise to localised "point" tenderness over posterior thoracic wall that would have its onset with *"sudden onset 10/10 left upper back/scapula pain – sharp – radiating to both jaws with nausea....radiation to occipital scalp"* and requiring 15 mg morphine to settle. In general it would not be safe to attribute such a presentation to musculoskeletal pain.

Non-traumatic causes of musculo-skeletal chest pain would generally be inflammatory or mechanical. Historical features suggesting a musculoskeletal cause include pain on specific postures or physical activities. Examination features such as the reproduction the patient's pain by either a movement or more specifically palpation over the structure that is the source of the pain is suggestive of a musculo-skeletal cause. In general inflammatory conditions would have a gradual onset and less severe pain, rather than the sudden '10/10' pain experienced by Mrs PIETZERIDES. Chest wall tenderness is a clinical sign that can lead to the diagnosis of musculoskeletal pain. In patients with acute chest pain, significant chest wall tenderness suggests that acute coronary syndrome and possibly other conditions are less likely but it does not effectively 'rule out' the diagnosis and implementing chest wall tenderness as an independent 'rule out' strategy in acute severe chest pain is unsafe and could lead to an unacceptably high rate of false negative (ie missed) diagnoses of serious conditions.

In his statement, Dr SMITH considers the patient to be at *"low risk of aortic dissection on the basis of the history, clinical examination and investigation results that did not demonstrate evidence of dissection. More specifically the chest x-ray examination did not show a widened mediastinum, the patient was not profoundly hyper or hypotensive and she was pain free within the Emergency Department apart from having initial mild pain.."*

However from the medical notes, of the three symptoms and signs in the 'classic triad' described in 'Clinical Prediction of Acute Aortic Dissection'[10] (ie aortic pain, widened mediastinum, pulse deficit), it is probable that 'aortic pain' is positive, widened mediastinum is negative (although the chest x-ray was not normal) and a pulse deficit or blood pressure difference was not examined for by hospital staff. If one applies the Risk Assessment Tool from the AHA guidelines in thoracic aortic disease,[20] Mrs PIETZERIDES had 2 characteristics of her pain are high-risk features. This alone put her at intermediate risk.

Dr SMITH also considered the patient to be at low risk because the patient was not *"profoundly hyper or hypotensive"* and was pain free within the ED, apart from having

mild initial pain. If 'profound' hypotension or hypertension had been present one would anticipate that Mrs PETZIERIDES would have been further investigated and/or admitted. Whilst hypertension is a risk factor for aortic dissection, blood pressure at the time of presentation is not a good discriminator for the presence or absence of acute aortic dissection. The IRAD[4] investigators found in the 289 Type A dissections examined approximately 40% were normotensive with a systolic blood pressure between 100-149 mmHg and approximately a quarter of Type B dissections were normotensive. Clearly an abnormal blood pressure is not a sensitive indicator of the presence of acute aortic dissection and it is incorrect to consider a patient at low risk on the basis of a relatively normal blood pressure. None of the articles reviewed utilised 'profound high or low pressure' at presentation to be an indicator of risk of acute aortic dissection.

Dr SMITH's clinical reasoning that put Mrs PIETZERIDES at low risk of aortic dissection was flawed on the basis of:

1. 30-40% of Type A dissections may have either normal chest x-ray or show no mediastinal widening or abnormal aortic outline. The absence of mediastinal abnormality cannot be used to 'rule out' aortic dissection
2. Hypertension or hypotension is not a sensitive marker of aortic dissection and 40% of Type A dissections are likely to be normotensive. A normal blood pressure cannot be used to 'rule out' aortic dissection.
3. The comment that she only had mild initial pain in the ED ignores the documented onset of the pain and the fact that dissection is a dynamic process that can occur in stages with pain initially settling, only to recur once dissection recommences.

If medical staff were aware of the limitations of the criteria upon which they based their decision making, the outcome may have been different.

The first ambulance crew to attend Mrs PETZIERIDES measured the blood pressure in both arms, as they were concerned about aortic dissection. They did not note a difference. As dissection is a dynamic process, it is possible that a blood pressure difference developed after this first assessment. Failure to look for a blood pressure difference at hospital suggests that the diagnosis was not considered and represents a missed opportunity to detect a 'classic' sign of aortic dissection and perhaps reconsider the diagnosis. The combination of typical aortic pain and a blood pressure differential has a high probability of dissection (of the order of >90%[10]).

It is possible that if a diagnosis of aortic dissection had been considered in hospital, either in the ED or in a ward if admitted, with appropriate investigation and treatment Mrs PETZIERIDES may have survived. Surgical survival rates are quoted at 75-90% for Type A dissections.[4,12]

It is also possible that if Mrs PETZIERIDES had been admitted hospital for observation or investigation of a suspected ACS she may have suffered rupture of her aorta in hospital. Given the time critical nature of this condition with regard to surgical repair and the specialised surgery required, she may have not survived.

In summary.

1. The care of Mrs PETZIERIDES has followed a conventional 'chest pain' pathway directed primarily at ruling out myocardial ischemia. Mrs PETZIERIDES was discharged with a diagnosis of musculoskeletal pain and a plan for further cardiology follow-up.

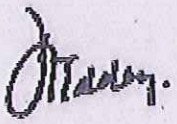
2. The first AV crew to attend Mrs PETZIERIDES considered the possibility of aortic dissection, examined for a blood pressure differential in the upper limbs and considered withholding aspirin because of this. Their clinical judgment is commendable, but it appears that their concerns were not communicated to clinical staff at Western Hospital because their case notes did not accompany the patient and possibly because the second (MICA) crew did not share or communicate their concerns. It is possible that if Western Hospital staff were made aware of AV paramedic concerns regarding aortic dissection, that they may have assessed Mrs PETZIERIDES as being of higher risk and undertaken diagnostic testing.

3. Despite obtaining a history of risk factors for aortic dissection and a description of the onset of pain that is highly suspicious, there is no indication that clinical staff at Western Hospital were either aware of AV paramedic concerns re aortic dissection or considered aortic dissection as a possibility themselves. They did not look for the classic clinical signs and once an ACS has been discounted the pain has been diagnosed as musculoskeletal.

4. Dr SMITH's statement indicates that he considered aortic dissection to be low risk based upon the history, examination and investigation results and the patient's lack of significant blood pressure abnormality. This risk assessment was based upon an incomplete examination, incorrect information and flawed reasoning. If medical staff had been aware of the limitations of the criteria upon which they based their decision making, the outcome may have been different.

5. Admission to hospital may have changed the outcome if the diagnosis of aortic dissection was considered prior to rupture, but if Mrs PETZIERIDES had ruptured her 'unsuspected' aortic dissection whilst in a short stay unit or ward bed, it is possible that she would not have survived.

I trust that this is of assistance with this matter.



Dr David Eddey *MB,BS, DipRACOG, DTM&H, Grad Cert Clin US, FACEM*

In the Coroners Court of Victoria at Melbourne

Court Reference: COR 2010 001571

Inquest into the death of **Constandia PETZIERIDES**

Attachment 2

Challenges to diagnosing aortic dissection in the ED

In order to diagnose aortic dissection four things need to occur. Firstly the clinician needs to be aware of the condition, however rare, as a cause for chest pain or one of the atypical presentations of aortic dissection. Secondly the clinician needs to take a history and perform a clinical examination in such a way as to get the appropriate information from the patient (ie they need to ask the right questions and examine the patient for specific signs). Thirdly they need to interpret the information from the point of view of risk of aortic dissection and lastly they need to perform appropriate investigations to diagnose or exclude the condition.

Because of the diverse manifestations of acute aortic dissection and the high chance of a fatal outcome if left untreated, clinicians must have a high index of suspicion for aortic dissection. Rapid recognition and treatment may improve survival and maximise recovery of peripheral malperfusion deficits such as stroke. Misdiagnosis as ACS may lead to inappropriate treatments – antiplatelet therapy and thrombolysis that may be lethal and coronary angiography. Delayed diagnosis may also delay antihypertensive therapy allowing propagation of the dissection and worsening prognosis.

The variable clinical manifestations of aortic dissection present a challenging differential diagnostic problem. Patients with aortic dissection may be diagnosed as having many other conditions, including myocardial infarction, pericarditis, pulmonary embolism, pneumothorax, pleurisy, ureteric colic, acute cholecystitis, biliary colic, musculoskeletal back pain, stroke, syncope, paraplegia, peripheral vascular disease and arterial embolism. Acute myocardial infarction is a common initial incorrect diagnosis, particularly in a patient with severe chest pain and an abnormal ECG.

If the Australian experience is similar to the American experience[30], a general ED seeing 50,000 patients per year might expect to see perhaps 5 acute aortic dissections per year. Clinical rarity may be a barrier to improving diagnostic accuracy and if this caseload is spread across the medical staff, each clinician might only experience a patient with this diagnosis every few years. Whatever the real figure, in all likelihood the number is quite low and an individual clinician's experience in diagnosing and managing acute aortic dissection is likely to be relatively modest.

If one considers clinicians working in low volume environments such as small rural hospital ED's supported by GP's on an on-call basis it is even less likely that an individual doctor or nurse will have had experience with aortic dissection.

Clinicians need to be able to consider the diagnosis a possibility in a particular patient amongst the 'background noise' of all patients that present with symptoms that could, but do not, represent acute aortic dissection. About 8% of all presentations to ED's the

UK are for the symptom of chest pain.[5] General metropolitan, suburban and major regional ED's in Victoria are likely to have a similar caseload. In addition to the very common presenting problem of chest pain, in order to accurately identify *all* cases of acute aortic dissection, the clinician must consider the diagnosis in patients with atypical presentations such as back pain, abdominal pain, syncope, or complaints related to a perfusion deficit including stroke, myocardial infarct, limb ischemia, and mesenteric ischemia.

Probably the most common diagnosis first considered in a patient presenting to the ED with chest pain is 'acute coronary syndrome' (ACS). Chest pain is a very common ED problem and whilst chest pain with ECG changes and a troponin rise is relatively 'straightforward' to deal with, chest pain may commonly be transient with no ECG changes or biomarker rise and not necessarily representative of the commencement of an immediately life threatening condition. In the assessment and risk stratification of patients thought to be at risk of ACS, patients are reasonably and relatively safely discharged if thought to be at low risk of an adverse event, even though coronary artery disease has not been excluded by coronary angiography. This is the basis of many chest pain pathways that focus on ischemic heart disease.

In contrast pain associated with aortic dissection represents the commencement of a highly lethal disease process and any pathway to diagnose aortic dissection in patients presenting with chest pain needs to be 100% sensitive, ie identify all patients with the disease around the time of presentation. It also needs to be relatively specific (ie exclude negatives) as it is possible that a significant percentage of patients with chest, abdominal or back pain of a non-aortic origin could be classified as intermediate or high risk, leading to over-testing. Because the accurate diagnosis or exclusion of aortic dissection requires an advanced imaging study, if every patient presenting with symptoms that might represent aortic dissection were imaged, both the monetary cost and radiation exposure would be prohibitive.

Unlike pathways designed specifically to manage or risk stratify patients for ischemic chest pain, there are no published Australian guidelines or pathways specifically for the assessment and diagnosis of patients with potential acute aortic dissection. Most 'chest pain pathways' focus on ischemic chest pain and although aortic dissection and other serious diagnoses may be mentioned in passing there is little in some guidelines to encourage a more detailed consideration of aortic dissection from the point of view of history, risk factors and anything more than the classic clinical findings.

For example the current Heart Foundation algorithm[31] "*Acute Coronary Syndromes Treatment Algorithm*" mentions 'suspected aortic dissection' only as a 'fine print' contraindication to thrombolysis for ST elevation myocardial infarction. Patients whose

symptoms are considered to be 'consistent' with ACS (acute coronary syndrome) but do not satisfy the criteria for reperfusion therapy then enter the risk stratification part of the algorithm. A patient such as Mrs PETZIERIDES is could easily be classified as intermediate risk (see Figure 3 below) and discharged home for a stress test and cardiology follow-up once troponin testing is normal. Whilst this is clearly labelled an ACS guideline and it possibly assumes that other causes have been excluded, there is little to encourage clinicians to consider alternative diagnoses, to review the diagnosis, or take a focussed history.

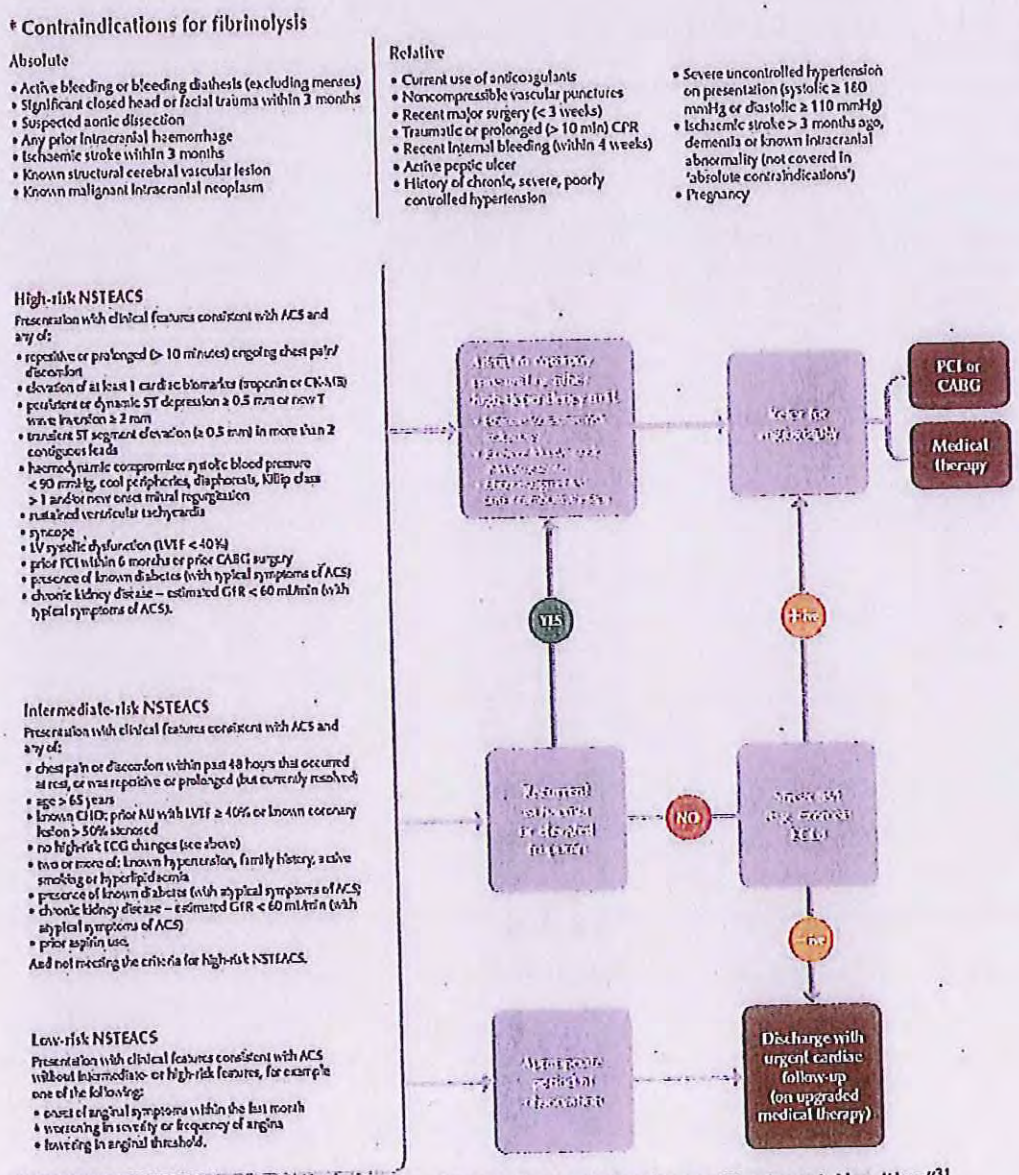


Figure 3. from National Heart Foundation "Acute Coronary Syndromes Treatment Algorithm"[31]

The NSW Health "NSW Chest Pain Pathway"[32] establishes mandatory requirements for all facilities with Emergency Departments to have and use a pathway that meets certain minimum standards. The chest pain pathway asks clinicians to consider aortic dissection or pulmonary embolism in patients with chest pain but no ST elevation, and lists back pain, hypertension, absent pulse and BP difference as features of this. This focus on the classic features of aortic dissection could encourage inexperienced clinicians to discount aortic dissection if these features are not present and ignores the circumstances where dissection involves the coronary arteries and produces ECG changes compatible with an ACS. There is no reference to symptoms of aortic dissection such as the quality and timing of the pain (see Figure 4 below).

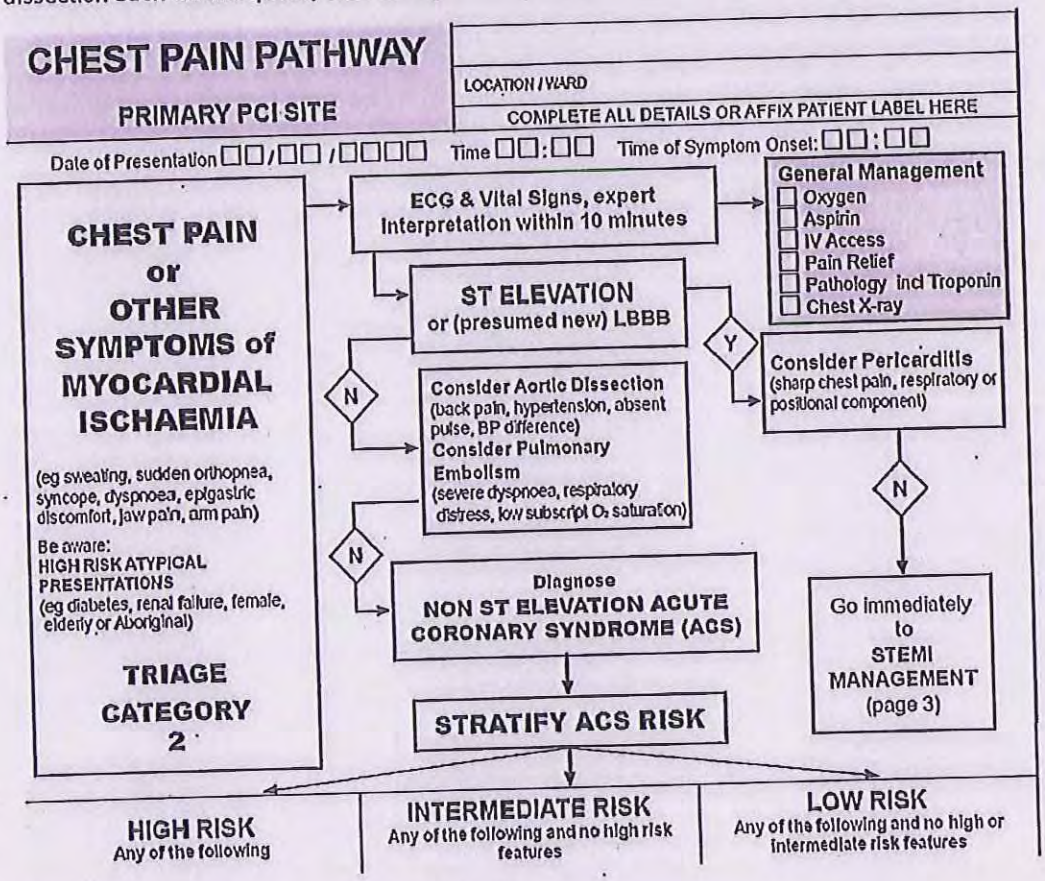


Figure 4 from NSW Health Chest Pain Pathway[32]

The Queensland Government "*Emergency Department Cardiac Chest Pain Risk Stratification Pathway*"[34] (Figure 5) advises users to "always consider other critical causes (eg PE, thoracic aortic dissection, abdominal aortic aneurysm" and goes on to continue to use the pathway unless there is a 'clear alternative diagnosis'. Once on this part of the pathway, there are no further reminders.

Queensland Government

Emergency Department

Cardiac Chest Pain Risk Stratification Pathway

Facility: _____

(Affix identification label here)

URN:

Family name:

Given name(s):

Address:

Date of birth: Sex: ☐M ☐F ☐I

Clinical Pathways *never replace clinical judgement*
Care outlined in this Pathway must be altered if it is not clinically appropriate for the individual patient
Timing of referral to cardiology / medical may vary for local circumstances

- *This pathway should be used for patients who have a complaint of chest discomfort (non-traumatic) or jaw, neck, shoulder, arm, back, or epigastric pain. Remember other atypical features (eg. diaphoresis, shortness of breath)*
- *Always consider other critical causes (e.g. PE, thoracic aortic dissection, abdominal aortic aneurysm)*

Medical Staff to perform risk stratification on the reverse of this form ⟶

Assessment	Date: / /	Time	Initial
1. Can you clearly diagnose non-cardiac chest pain as an alternative diagnosis? ☐ Yes - clear alternative diagnosis. Stop pathway *(state reason)*: _____ ☐ No - use this pathway and perform risk stratification over page			
2. Initial observations attended			

Figure 5 from Queensland Government *"ED Cardiac Chest Pain Risk Stratification Pathway"*[34]

Another pathway, from SA Health ICCnet (Integrated Cardiovascular clinical Network): *"ICCnet SA management of Chest Pain/Suspected Acute Coronary Syndrome"*[33] does not consider aortic dissection at all. The National Institute for Health and Clinical Excellence (United Kingdom NHS) guideline[35] *"Chest pain of recent onset: Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin"* does not specifically mention aortic dissection or its features, although it advises clinicians to consider other acute conditions, firstly life-threatening conditions.

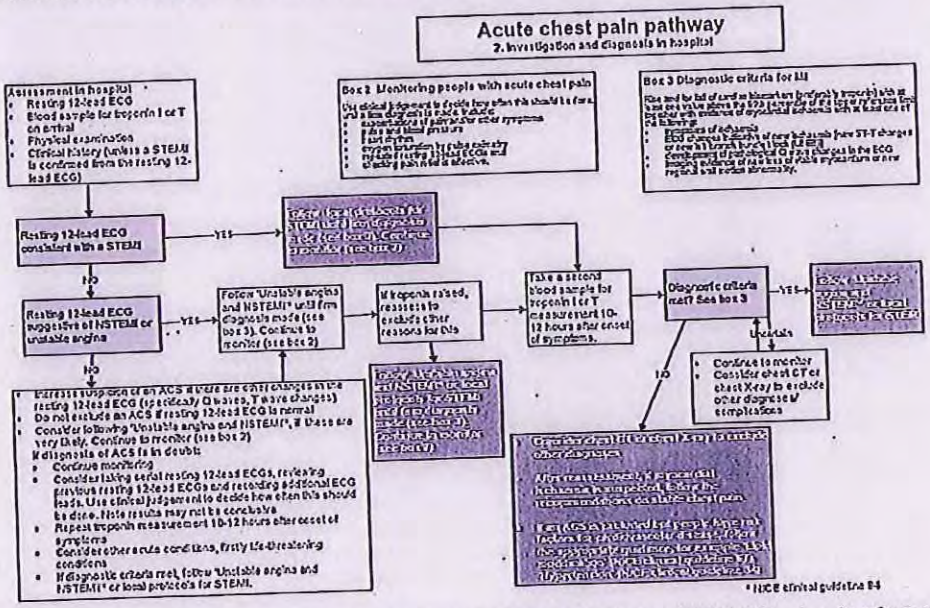


Figure 6 from National Institute for Health and Clinical Excellence (NHS) *"Chest pain of recent onset"*[35]

Whilst such guidelines are directed managing the huge numbers of patients presenting with chest pain that could represent an acute coronary syndrome, some are somewhat misleadingly called a 'chest pain pathway'. In reality they generally direct patients down the cardiac risk stratification route if the patient's symptoms are considered to represent a *"presentation with clinical features consistent with ACS and any of....."* Severe chest pain will obviously be considered by clinicians to be a clinical feature consistent with ACS.

It would also be reasonable to suspect that there may be a greater reliance by staff of small health services on these pathways to manage chest pain presentations, and that their level of suspicion for aortic dissection may be lower than clinicians who deal with chest pain emergencies on a regular basis.

The fact that a patient is on an 'approved' chest pain pathway may provide false reassurance to inexperienced or unwary clinicians.

Once a patient is on a 'pathway' and particularly if the patient remains pain free and is handed over to other staff or transferred to an observation unit, there may be no further history taking, re-examination of the patient or re-consideration of the diagnosis. If the patient's subsequent biomarkers and ECG's are unchanged the patient may well be discharged without further medical review.

Conditions within the ED and hospital with regard to patient flow (ie discharge within 4 hours) and workload make the use of pathways for common conditions such as chest pain an attractive way of managing this, but a focus on 'ticking the boxes' of a pathway and moving the patient on is not necessarily conducive to identifying the occasional stable patient who nevertheless has a potentially immediately life threatening cause for their presentation.

The main challenge in managing acute aortic dissection is to suspect and diagnose the condition as early as possible. There is evidence[24] to suggest that the quality of history taking is poor and my personal experience as a supervising ED consultant with junior and middle level medical staff taking a history from patients with chest pain would anecdotally support this. In my experience there is also a tendency for clinicians to discount the possibility of aortic dissection if the classic signs of blood pressure differential and widened mediastinum are absent. This is probably not confined to ED staff, as many of these patients are likely to be admitted under medical or cardiology units with ischemic chest pain as a working diagnosis.

From a practical point of view if these patients are to be identified and investigated appropriately without massively increasing cost and the use of CT, good quality clinical

history and examination are paramount. In order to do this the clinician must first consider aortic dissection as a possibility. This is possibly the biggest barrier to identifying the condition.

The American College of Cardiology and American Heart Association Task Force on Practice Guidelines for the management of thoracic aortic disease[20], introduced an evaluation and management algorithm for patients presenting with symptoms possibly attributed to dissections, such as chest, back, or abdominal pain or syncope.

It classifies patients into low, intermediate, or high-risk categories on the basis of a targeted history to assess dissection risk factors and the physical examination. This algorithm was retrospectively tested using data on 2538 dissection patients presenting to centres participating in the international registry of aortic dissection. Results suggested that 97.5% of these patients would have been identified by a risk factor or classic symptom or sign of an acute dissection.[30]

The clinical algorithm and risk score utilised in this study is at Figure 7 below. It attempts to identify patients at risk of aortic dissection by a 'bedside risk assessment'. In particular it asks clinicians to perform a focussed bedside assessment to identify:

1. High-risk conditions (eg Marfan syndrome, known aortic valve disease, known thoracic aortic aneurysm).
2. High-risk features (Chest, back or abdominal pain of abrupt onset, severe intensity and ripping or tearing in quality). Notably sharp chest pain has not been utilised.
3. High risk examination features (Pulse deficit, systolic blood pressure difference, focal neurologic deficit with associated pain, new aortic regurgitation murmur in conjunction with pain, hypotension or shocked state)

A score out of three for the number of categories in which the patient has positive findings is given.

- For patients with no features (ie score = 0) clinical evaluation is undertaken as indicated by the presentation. Aortic imaging is expedited if there is unexplained hypotension of a widened mediastinum on plain chest x-ray.
- For patients with a positive feature in one of the three categories (ie score = 1) the patient is assessed with ECG, chest x-ray and may undergo expedited aortic imaging unless another diagnosis is confirmed.
- Patients with positive features in two or more categories (score = 2-3) undergo expedited aortic imaging and surgical/cardiological consultation.

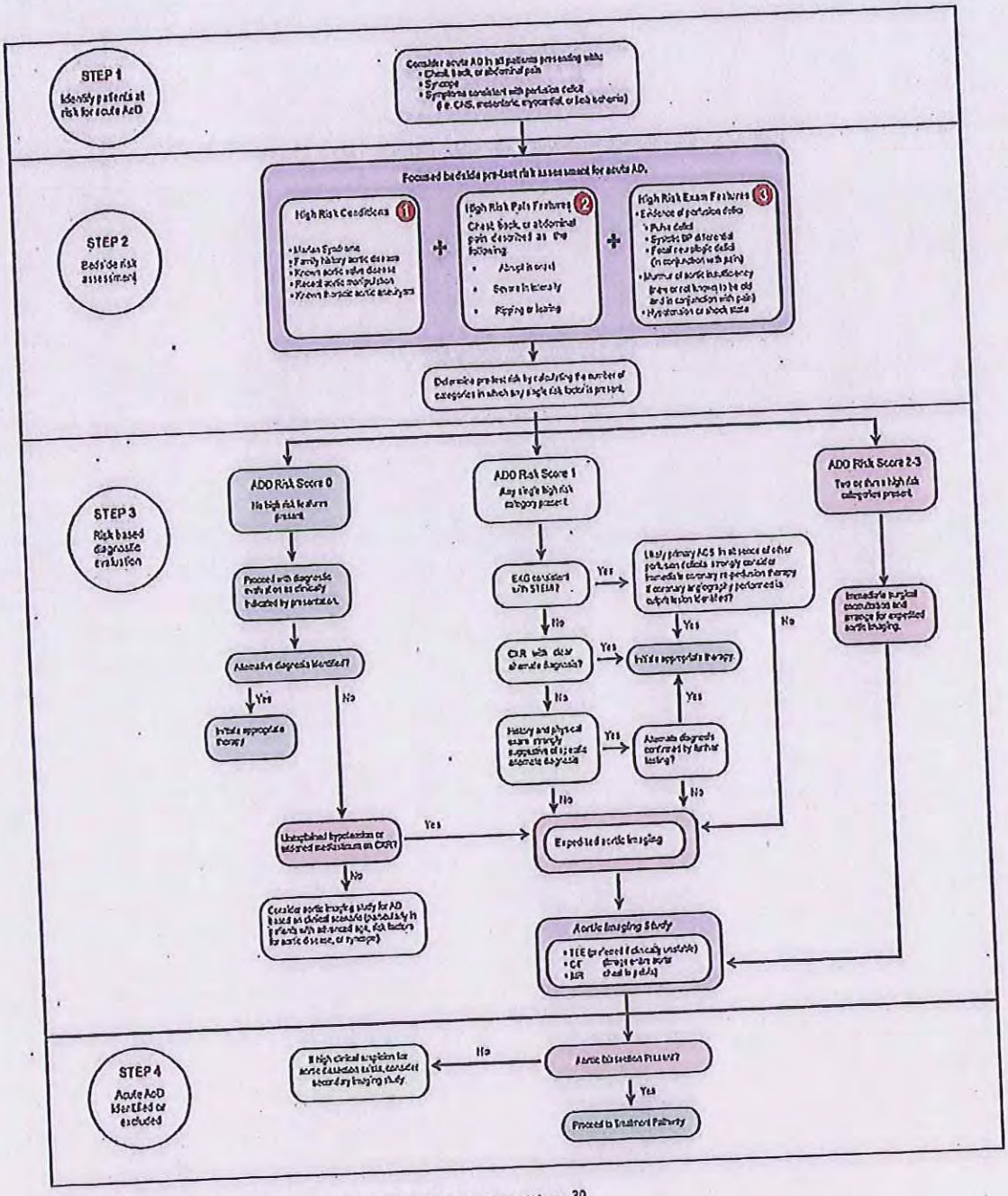


Figure 7 Aortic Dissection Detection Risk Score algorithm.[30]

This tool has not been prospectively validated in a clinical setting. This would be difficult due to very low case numbers and one might anticipate limitations in an undifferentiated patient population. In view of the relative rarity of acute aortic dissection, which may result in misdiagnosis or delayed diagnosis, the application of such a 'risk score' has the potential to at least draw the necessary clinical attention to

the possibility of acute aortic dissection. If the assessment of predisposing conditions for dissection, along with the signs and symptoms, was routinely incorporated into chest pain assessment guidelines it might lead to improved assessment of patients with chest pain and risk factors for aortic dissection.

In summary, impediments to diagnosing aortic dissection in the pre-hospital, Emergency Department and inpatient ward settings may arise because of:

1. Clinical rarity and failure to think of the condition.
2. Poor quality history taking and failure to clarify the characteristics of the pain.
3. A perception that aortic dissection is unlikely if the classic signs of blood pressure differential, pulse deficit and widened mediastinum are absent.
4. An emphasis on much higher volume cardiac chest pain and 'chest pain' pathways that focus primarily on acute coronary syndromes without encouraging re-evaluation of the patient and diagnosis.
5. Conditions within ED's and work practices that encourage patients to be moved on within short time frames.

In the Coroners Court of
Victoria at Melbourne

Court Reference: COR 2010 001571

Inquest into the death of **Constandia PETZIERIDES**

Attachment 3

Advice as to best practice in ED with regard to suspected aortic dissections

The goal of these recommendations is to improve physician recognition and facilitate prompt diagnostic testing and management of those at risk. Best practice in ED's is confined to the initial diagnosis and early management of aortic dissection. Definitive care is not the realm of the ED.

In order to diagnose aortic dissection three things need to occur. Firstly the clinician needs to be aware of the condition, however rare, as a cause for chest pain or one of the atypical presentations of aortic dissection (ie the clinician needs to think of it!).

Secondly the clinician needs to take a history and perform a clinical examination and investigations in such a way as to get the appropriate information from the patient (ie they need to ask the right questions and examine the patient for specific signs).

Thirdly the clinician must interpret the information gained from the point of view of risk of aortic dissection or other conditions. This includes an appreciation of the limitations of history, clinical signs and simple investigations with regard to their ability to 'rule in' or 'rule out' the diagnosis.

As most patients with aortic dissection present with sudden onset severe chest pain and the biggest improvements in identifying patients with aortic dissection early are likely to be made from focussing on these patients. Significantly smaller numbers of patients would present with atypical signs and symptoms.

Best practice in ED's with regard to improving the diagnosis and survival of patients with aortic dissection could include

1. Improve clinician (doctor, nurse, paramedic) awareness of aortic dissection by targeted education.
2. Improve clinician history taking. In particular, in addition to routine clinical history taking, patients presenting with severe chest pain should be specifically questioned regarding:
    a. The presence of risk factors, particularly hypertension, family history, recent procedures and Marfan syndrome.
    b. The onset of pain, in particular its severity at onset and the time taken to reach maximal severity.
    c. The character of the pain.
    d. Location and radiation of pain and changes in location.
    e. Associated features such as syncope, neurological deficit, back or abdominal pain.

3. Improve clinical examination. In particular, in addition to routine clinical examination, patients presenting with severe chest pain should receive focused examination of:
    a. Blood pressure for differential (>20mmHg) between upper limbs.
    b. Major pulses to detect pulse deficit (ie absence). This includes carotid arteries, radial arteries and femoral arteries.
    c. Jugular venous pressure and blood pressure for pulsus paradoxus to detect evidence of cardiac tamponade.
    d. Heart sounds to detect aortic regurgitation murmur.
    e. The abdomen for evidence of visceral ischemia
    f. The central nervous system for evidence of neurological deficit.
4. Improve investigation utilisation and interpretation. In particular, in addition to routine investigations such as ECG, patients with acute severe chest pain should undergo plain chest x-ray to examine for:
    a. mediastinal widening and aortic outline.
    b. Other findings such as pleural effusion (?haemothorax) and significant cardiomegaly (? pericardial effusion) are important findings if present.
5. Clinical education to clarify the facts around the reliability of symptoms, signs and investigations with regard to their ability to rule in or rule out the diagnosis.
6. Patients with high-risk features should be discussed with a senior clinician with a view to expediting further investigation and management. Consider admitting all patients with an episode of unexplained severe chest pain.
7. Clinical education with regard to risk assessment and early referral of patients with high risk features to senior clinicians, specialty consultation and transfer when necessary. A risk assessment tool could be utilised, eg AHA guidelines.[20]

Risk Assessment	
High Risk conditions	Marfan syndrome Family history of aortic disease Known aortic valve disease Recent aortic manipulation Known thoracic aortic aneurysm
High Risk Features	Chest, back or abdominal pain with any of the following characteristics • Abrupt onset • Severe intensity • Ripping or tearing quality
High Risk examination features	Evidence of perfusion deficit • Pulse deficit • Systolic blood pressure differential • Focal neurological deficit Aortic regurgitation murmur and pain Hypotension or shock state

8. Further investigation should include urgent CT aortogram if the patient is clinically stable (or MRI aortogram in the patient is suitable). In unstable patients trans-oesophageal echocardiography should be undertaken in a critical care environment.
9. In patients with diagnosed aortic dissection, urgent consultation with an appropriate specialist service must take place – ie cardiology, cardiothoracic surgery.
10. Immediate care should be directed at resuscitation, if required, and decreasing aortic wall stress by controlling heart rate and blood pressure. Care should be taken not to induce reflex tachycardia. In the presence of acute aortic regurgitation care should be exercised if suppressing a compensatory reflex tachycardia.
11. Definitive care should be undertaken in a centre with appropriate expertise and relevant specialties including cardiology, cardiothoracic surgery, vascular surgery and intensive care. Transfer should be arranged through adult retrieval Victoria.
12. Consider amending current 'chest pain pathways' to 'cardiac chest pain' pathways if specifically focussed upon ACS. This could assist in reducing the 'reassurance' obtained by placing an undifferentiated chest pain patient on such a pathway.
13. Modify 'chest pain' guidelines and pathways to include specific information regarding features of the history and the limitations of clinical findings and investigations in ruling out aortic dissection, and to encourage consideration of aortic dissection in patients with high-risk features.
14. Develop a severe chest pain guideline (ED/Cardiology/Cardiothoracics/Radiology) and screening tool.
15. Develop a hospital transfer guideline or policy to ensure that high risk patients receive expedited transfer (particularly from smaller and rural health services) to a centre able to provide the required level of definitive care recognising that his may result in some patients, who ultimately are proven not to have aortic dissection, being transferred.

With regard to the first 10 points above I have appended a sample information sheet that could represent how this would look. (Appendix 1)

Appendix 1:

Recognising Aortic dissection

1. Acute aortic dissection is relatively rare, but failure to consider the diagnosis may have catastrophic consequences with untreated mortality rates of 90% or more.
2. **Focused history taking for risk factors and typical symptoms and a thorough clinical examination are the keys to identifying the majority of these patients early.**
3. Risk factors include hypertension, atherosclerosis, known aortic aneurysm, known aortic valve disease (eg bicuspid aortic valve), family history of aortic disease, recent aortic surgery or catheterisation and Marfan syndrome. The occurrence of chest pain in a patient with Marfan syndrome should mandate investigation of the aorta.
4. The 'classic presentation' of aortic dissection (ie 'tearing' interscapular pain, blood pressure difference in the upper limbs and a widened mediastinum on chest x-ray) occurs in only about one third of patients with acute aortic dissection.
5. Most (90%) patients with acute aortic dissection present with 'immediate' onset of very severe chest pain ('worst ever' in 90%) that is at its worst from the outset.
6. Pain from aortic dissection is classically described (50% of patients) as tearing or ripping. Pain is more commonly (64%) described as sharp or stabbing, although this is less specific.
7. Interscapular pain, posterior chest or back pain is more common in distal dissections but frequently (>70%) patients with proximal dissections experience anterior chest pain that can radiate (27%) to the neck, throat or jaw.
8. Pain from aortic dissection may completely resolve and should not lead clinicians to discount the diagnosis, particularly if risk factors and high-risk features exist. Dissection may occur in stages, with recurrence of the pain when the dissection recommences.
9. As dissection progresses, pain may migrate. Involvement of aortic branches may produce stroke, spinal cord lesion, renal or gonadal pain, abdominal pain and poor perfusion of limbs. Due to intermittent occlusion these effects may be transient or recurrent.
10. **Normal blood pressure on presentation should not be used to 'rule out' the diagnosis of aortic dissection clinically.** 40% of proximal dissections and 25% of distal dissections may be normotensive on presentation. 70% of distal dissections and 35% of proximal dissections may be hypertensive and up to 25% of proximal dissections and 5% of distal dissections may present shocked.
11. **Blood pressure differentials or pulse deficits are predictive of aortic dissection, but are not sensitive enough to be used to 'rule out' the diagnosis.** Pulse deficits or blood pressure differences may be transient. Less than 20-40% of proximal dissections and 10-20% of distal dissections may have a pulse deficit.
12. **A new aortic regurgitation murmur in the setting of severe chest pain**
13. There are no diagnostic ECG changes of aortic dissection. A normal or non-specific ECG in the presence of severe chest pain should raise suspicions of aortic dissection. Commonly there may be non-specific ST-T changes or features of LVH. The presence of frank ischemia on the ECG may indicate involvement of the coronary arteries in the process and should not necessarily rule out the diagnosis of aortic dissection.

14. A widened mediastinum or abnormal aortic outline are 'classic' chest x-ray findings in aortic dissection. 47-63% of proximal dissections and 50-60% of distal dissections may have these findings, but these features may be absent in 30-40% of dissections. **The absence of 'classic' chest x-ray findings cannot be used to rule out the diagnosis of aortic dissection.**
15. Currently there are no validated laboratory tests to rule out aortic dissection. **D-dimer may be elevated in aortic dissection, but a normal D-dimer is not sensitive enough to use to 'rule out' aortic dissection.** Troponin may be elevated in cases where there is myocardial ischemia associated with aortic dissection, and care should be taken not to discount the diagnosis of aortic dissection solely on this basis.
16. Aortic dissection may present atypically as syncope and chest pain, neurological symptoms (stroke or spinal cord deficits) and pain or abdominal pain. **Neurological symptoms in the setting of chest pain should mandate consideration of aortic dissection.**
17. Aortic dissection may present painlessly with or without peripheral symptoms and signs. This is more likely to occur in chronic aortic dissection.

Assessment of Patients with Acute Severe Chest Pain

Clinical History

In addition to routine clinical history taking, patients presenting with severe chest pain should be specifically questioned regarding:

1. Presence of risk factors, particularly hypertension, family history, recent procedures and Marfan syndrome.
2. The onset of pain, in particular its severity at onset and the time taken to reach maximal severity.
3. The character of the pain.
4. Location and radiation of pain and changes in location
5. Associated features such as syncope, neurological deficit, back or abdominal pain.

Clinical Examination

In addition to the routine clinical examination, patients presenting with severe chest pain should receive examination of:

1. Blood pressure for differential (>20mmHg) between upper limbs.
2. Major pulses to detect pulse deficit (ie absence). This includes carotid arteries, radial arteries and femoral arteries.
3. Jugular venous pressure and blood pressure for pulsus paradoxus to detect evidence of early tamponade.
4. The heart to detect aortic regurgitation murmur and evidence of heart failure.
5. The abdomen for evidence of visceral ischemia
6. The central nervous system for evidence of neurological deficit.

Investigations

In addition to routine investigations such as ECG, patients with acute severe chest pain should undergo plain chest x-ray to examine for mediastinal widening and aortic outline. Other findings such as pleural effusion (?haemothorax) and significant cardiomegaly (? pericardial effusion) are important findings if present.

Risk assessment

Based on the clinical history, examination and basic investigations an assessment of the risk of aortic dissection should be made to guide further management.

Patients with high-risk features should be discussed with a senior clinician with a view to expediting further investigation and management. Consider admitting all patients with an episode of unexplained severe chest pain.

Patients with at least one positive finding in any one of the three risk categories can be considered to be at intermediate risk. Patients with at least one positive finding in two or more of the risk categories should be considered to be at high risk.

Risk Assessment	
High Risk conditions	Marfan syndrome Family history of aortic disease Known aortic valve disease Recent aortic manipulation Known thoracic aortic aneurysm
High Risk Features	Chest, back or abdominal pain with any of the following characteristics • Abrupt onset • Severe intensity • Ripping or tearing quality
High Risk examination features	Evidence of perfusion deficit • Pulse deficit • Systolic blood pressure differential • Focal neurological deficit Aortic regurgitation murmur and pain Hypotension or shock state

Further investigations

If indicated, further investigation should include CT aortogram, MRI aortogram or Trans-oesophageal echocardiography.

Immediate Care

Immediate care should be directed at resuscitation if required and decreasing aortic wall stress by controlling heart rate and blood pressure. Care should be taken not to induce reflex tachycardia. In the presence of acute aortic regurgitation care should be exercised if suppressing a compensatory reflex tachycardia

Consultation

In patients with diagnosed aortic dissection, urgent consultation with an appropriate specialist service must take place – ie cardiology, cardiothoracic surgery

Definitive Care

This should be undertaken in a centre with appropriate expertise and relevant specialties including cardiology, cardiothoracic surgery, vascular surgery and intensive care. Transfer should be arranged through adult retrieval Victoria.

In the Coroners Court of Victoria at Melbourne

Court Reference: COR 2010 001571

Inquest into the death of **Constandia PETZIERIDES**

Attachment 4

HOSPITAL	TITLE	FIRST NAME	SURNAME	POSITION DESCRIPTION	Atending
Coroners Court of Victoria	Coroner	Paresa	Spanos	Coroner	Yes
Alfred Health	Dr	Helen	Stergiou	Deputy Director Emergency Department	Yes
Austin Health	Dr	Fergus	Kerr	Director Emergency Department	Yes
Austin Health	Dr	Simon	Judkins	Clinical Director Emergency Department	Yes and Dr Michael Yeoh
Box Hill Hospital	Dr	Yvette	Kozielski	Medico Legal Consultant	Yes and Dr Andrea McLean
Maroondah Hospital	Dr	Peter	Archer	Director Emergency Department	
Angliss Hospital	Dr	Graeme	Thomson	Director Emergency Department	Yes
Royal Melbourne Hospital	Dr	Steve	Pincus	Acting Director Emergency Department	Yes and Dr Peter Ritchie
Northern Hospital	Dr	Shyaman	Menon	Director Emergency Department	Yes
Northern Hospital	Dr	James	Hayes	Emergency Physician in ED	Yes
Frankston Hospital	Dr	Helen	Hewitt	Director Emergency Department	Yes
Frankston Hospital	Dr	Sherene	Devanesen	Chief Executive Officer	
Dandenong Hospital	Professor	George	Braitberg	Director Emergency Department	
Dandenong Hospital	Dr	Neil	Goldie	Director Emergency Department	Yes
Monash Medical Centre	Dr	Tony	Kambourakis	Director Emergency Department	Yes
St Vincents Health	Dr	Jonty	Karro	Emergency Physician	
Werribee Mercy Hospital	Dr	John	Pasco	Chief Medical Officer	Yes - Michael Bryant

Footscray Hospital	Dr	Anne-Maree	Kelly	Emergency Physician in ED	Yes
Sunshine Hospital	Dr	Peter	Ritchie	Director Emergency Department	Yes
Eastern Health	Dr	Robyn	Parker	Emergency physician	Yes
Coroners Court of Victoria	Dr	Sandra	Neate	Emergency physician	Yes
Coroners Court of Victoria	Dr	George	Jelinek	Emergency physician	Yes
Coroners Court of Victoria	Mr	John	Milroy	Clinical Nurse	Yes
Coroners Court of Victoria	Ms	Ruth	Bergman	Senior Clinical Nurse	Yes
Coroners Court of Victoria	Leading Senior Constable	Tania	Cristiano	Police Coronial Support Unit/ Coroner's Assistant	Yes